MEDICAL LIBRARY

FEB 14 1948

VOLUME 47 NUMBER 1

ARCHIVES OF

OTOLARYNGOLOGY

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

WESTLEY M. HUNT, New York
RALPH A. FENTON, Portland Ore.
ERNEST M. SEYDELL, Wichita, Kan.
CHEVALIER JACKSON, Philadelphia
ROBERT L. GOODALE, Boston
J. MACKENZIE BROWN, Los Angeles
CARL H. McCASKEY, Indianapolis

JANUARY 1948

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $10.00

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1948, BY THE AMERICAN MEDICAL ASSOCIATION

ARCHIVES OF OTOLARYNGOLOGY

VOLUME 47 JANUARY 1948 NUMBER 1

COPYRIGHT, 1948, BY THE AMERICAN MEDICAL ASSOCIATION

THE QUESTION OF PROTHROMBINOPENIC HEMORRHAGE FROM POST-TONSILLECTOMY USE OF CHEWING GUM CONTAINING ACETYLSALICYLIC ACID

GEORGE S. LIVINGSTON, M.D.
CHICAGO
AND
EDWARD R. NEARY, M.D.
NEWARK, N. J.

THE PROTHROMBINOPENIC effect of acetylsalicylic acid and the salicylates and the protective action of vitamin K, administered with these drugs, have been demonstrated by a number of investigators.[1] The clinical significance of these findings has presented an importunate problem for study, particularly with reference to whether in man salicylate-induced prothrombinopenia is of sufficient magnitude to cause hemorrhage. In this regard, Quick [2] has stated that the blood prothrombin must be reduced below 20 per cent of normal before there is danger of hemorrhage and that salicylates, even when given in massive doses, do not cause the prothrombin to fall to the hemorrhagic level.

Quick's view seems to have been confirmed by the more recently reported results of extensive studies made by the Armed Forces, in which large numbers of patients with rheumatic fever were treated with massive doses of salicylates. Coombs and collaborators [3] followed

From the Otho S. A. Sprague Memorial Institute Laboratory, Children's Memorial Hospital, Chicago (Dr. Livingston), and the Medical Department, White Laboratories, Inc., Newark, N. J. (Dr. Neary).

1. Link, K. P.; Overman, R. S.; Sullivan, W. R.; Huebner, C. F., and Scheel, L. D.: Studies on the Hemorrhagic Sweet Clover Disease: XI. Hypoprothrombinemia in the Rat Induced by Salicylic Acid, J. Biol. Chem. **147**:463 (Feb.) 1943. Meyer, O. O., and Howard, B.: Production of Hypoprothrombinemia and Hypocoagulability of the Blood with Salicylates, Proc. Soc. Exper. Biol. & Med. **53**: 234 (June) 1943. Shapiro, S.; Redish, M. H., and Campbell, H. A.: Studies on Prothrombin: IV. The Prothrombinopenic Effect of Salicylate in Man, ibid. **53**:251 (June) 1943.

2. Quick, A. J.; Salicylates and Hemorrhage, J.A.M.A. **126**:1167 (Dec. 30) 1944.

3. Coombs, F. S.; Warren, H. A., and Higley, C. S.: Toxicity of Salicylates, J. Lab & Clin. Med. **30**:378 (April) 1945.

salicylate blood levels in 48 Army patients with acute rheumatic fever during oral and intravenous administration of daily doses of 10 to 16 Gm. of salicylate. In discussing the effect produced on blood prothrombin, the authors state that no significant effect was noted even when blood salicylate levels reached approximately 35 mg. per hundred cubic centimeters. Butt and associates [4] also observed the prothrombin content of the blood during administration of large amounts of sodium salicylate. Elevation of prothrombin time was noted in a large percentage of cases only when a dose of 10 Gm. or more of sodium salicylate was employed. According to these investigators, even with this dose the increase of prothrombin time was not pronounced, and at no time in any case was a hemorrhagic manifestation noted. Citing such experiences, Higley [5] has expressed the opinion that, although salicylates do cause slight prolongation of the prothrombin time, this is not clinically significant with daily doses up to 10 Gm. and hence the routine addition of vitamin K to the acetylsalicylic acid is not warranted.

Although the consensus of clinical investigators now seems to be that large doses and continued administration of salicylates may decrease blood prothrombin but not to an extent sufficient to cause hemorrhage, it has been suggested that a common cause of late post-tonsillectomy hemorrhage is prothrombinopenia induced with acetylsalicylic acid. Singer [6] has related the secondary hemorrhage occurring in tonsillectomized patients to the postoperative use of acetylsalicylic acid, essentially on the ground that late hemorrhage is a relatively more frequent post-tonsillectomy complication in the United States, where administration of acetylsalicylic acid is a postoperative routine, than in Europe, where the drug is never used as a postoperative analgesic. The fallacy of Singer's reasoning has been commented on by Higley.[5] Nevertheless, Neivert,[7] also, has suggested that one of the most important causes of late post-tonsillectomy hemorrhage is a reduction of blood prothrombin brought about by the use of acetylsalicylic acid or salicylates. He reported that five to seven days after operation significant prothrombino-

4. Butt, H. R.; Leake, W. H.; Solley, R. F.; Griffith, G. C.; Huntington, R. W., and Montgomery, H.: Studies in Rheumatic Fever: I. The Physiologic Effect of Sodium Salicylate on the Human Being, with Particular Reference to the Prothrombin Level of the Blood and the Effect on Hepatic Parenchyma, J.A.M.A. **128**:1195 (Aug. 25) 1945.

5. Higley, C. S.: Use of Salicylates in Rheumatic Fever—Mixture of Aspirin and Vitamin K Unwarranted, J.A.M.A. **129**:1123 (Dec. 15) 1945.

6. Singer, R.: Acetylsalicylic Acid: A Probable Cause for Secondary Post-Tonsillectomy Hemorrhage, Arch. Otolaryng. **42**:19 (July) 1945.

7. Neivert, H.: Late Secondary Tonsillar Hemorrhage: I. Studies of Prothrombin and Vitamin K, Arch. Otolaryng. **42**:14 (July) 1945.

penia occurred in a majority of tonsillectomized patients to whom acetylsalicylic acid was administered in tablet and chewing gum form; that postoperative prothrombinopenia did not occur in patients who received both acetylsalicylic acid and synthetic vitamin K, and that in the latter patients the incidence of secondary hemorrhage was relatively low. However, in Neivert's report there is no direct correlation between decreased blood prothrombin and the occurence of late post-tonsillectomy hemorrhage.

PRESENT STUDY

The purpose of the present study was to determine whether the routine post-tonsillectomy use of a chewing gum containing acetylsalicylic acid induces prothrombinopenja and, if so, whether secondary postoperative hemorrhage is etiologically related to the lowered blood prothrombin.

The tonsillectomized patients who served as subjects of study were children less than 13 years of age, all of whom were ward patients in whom tonsillectomy had been clearly indicated. Only the records of those who gave evidence of having been fully cooperative during the course of study, a total of 45 patients, were regarded as satisfactory for this report.

An estimation of blood prothrombin was made by Kato's [8] method for every patient on the day prior to operation, on the afternoon following operation in the morning and one week after operation. Kato's technic is a micromethod in which small quantities of oxalated whole capillary blood are used instead of the larger amounts of venous plasma used in other methods, which require venipuncture. Obviously, Kato's method is especially practicable with young children. The average normal prothrombin time measured by this method is 20 seconds with a deviation of ±2.

On the day following operation our patients received a supply of medicated chewing gum with specific directions calling for daily chewing of a total of 5 to 6 gum tablets until they returned for examination and final estimation of prothrombin on the seventh postoperative day. One group (29 patients) received chewing gum tablets containing approximately 0.23 Gm. (3½ grains) of acetylsalicylic acid; a second group (16 patients) received gum tablets containing the same quantity of acetylsalicylic acid together with 0.5 mg. of menadione, as controls.[9] This amount of menadione was believed to be more than ample for counteracting whatever prothrombinopenic effect the contained acetylsalicylic acid might induce, since Shapiro [10] has estimated that approximately 1 mg. of menadione will counteract the prothrombin-lowering action of 1 Gm. of acetylsalicylic acid.

---

8. Kato, K.: Micro-Prothrombin Test with Capillary Whole Blood: A Modification of Quick's Quantitative Method, Am. J. Clin. Path. **10**:147 (Feb.) 1940.

9. The medicated chewing gum tablets were supplied by White Laboratories, Inc., Newark, N. J.

10. Shapiro, S.: Studies on Prothrombin: The Effect of Synthetic Vitamin K on the Prothrombinopenia Induced by Salicylate in Man, J.A.M.A. **125**:546 (June 24) 1944.

TABLE 1.—*Prothrombin Values of Blood Samples Obtained from Tonsillectomized Children Who Were Treated with Chewing Gum Containing Acetylsalicylic Acid*

Patient	Prothrombin Time, per Cent of Normal *		
	Preoperative Test	Postoperative Tests	
		4-6 Hours	1 Week
1	100	92	90
2	81	89	100
3	88	89	86
4	97	87	96
5	87	80	70
6	100	75	76
7	93	81	80
8	100	80	80
9	81	89	96
10	96	56	100
11	74	65	100
12	78	66	100
13	91	72	80
14	89	72	84
15	115	86	92
16	104	61	107
17	124	80	102
18	143	93	82
19	108	109	104
20	93	95	87
21	75	91	91
22 †	98	111	94
23 †	98	114	128
24	105	96	122
25	100	125	128
26	102	89	115
27	110	100	111
28	104	95	100
29	106	93	97

* The blood concentration of prothrombin is inversely proportional to the clotting time of the blood in this test.[8]

† This patient experienced a secondary post-tonsillectomy hemorrhage.

TABLE 2.—*Prothrombin Values of Blood Samples Obtained from Tonsillectomized Children Who Received Chewing Gum Containing Acetylsalicylic Acid and Menadione*

Patient	Prothrombin Time, per Cent of Normal *		
	Preoperative Test	Postoperative Tests	
		4-6 Hours	1 Week
30	123	95	116
31 †	73	68	104
32	89	53	117
33	83	54	101
34	122	108	106
35	84	97	104
36	91	121	83
37	89	104	94
38	112	91	83
39	96	102	121
40	104	106	117
41	104	110	138
42	104	117	128
43	96	93	107
44	107	96	97
45	82	89	120

* The blood concentration of prothrombin is inversely proportional to the clotting time of the blood in this test.[8]

† This patient experienced a secondary post-tonsillectomy hemorrhage.

FINDINGS

The results of our study of the prothrombin levels of both the treated and the control group of patients are summarized in tables 1 and 2. The first two columns of tabulated figures represent premedication estimations of blood prothrombin. The last column of figures, representing prothrombin values after one week's chewing of the medicated gum tablets, should reflect any significant effect which the use of acetylsalicylic acid gum tablets or acetylsalicylic acid–menadione gum tablets may have exerted.

The tabulated results make it apparent, first, that the normal blood prothrombin time may vary widely and, secondly, that no specific variations of prothrombin time may be ascribed to the use of either acetylsalicylic acid or menadione–acetylsalicylic acid chewing gum. The postmedication prothrombin values indicate that no significant decrease of prothrombin results from the post-tonsillectomy use of chewing gum containing acetylsalicylic acid and, conversely, that the addition of menadione to the acetylsalicylic acid contained in the gum is neither necessary nor advantageous.

Late Post-Tonsillectomy Hemorrhage.—In the group of 29 tonsillectomized patients treated with chewing gum containing acetylsalicylic acid there were 2 (nos. 22 and 23) in whom secondary postoperative hemorrhage occured. In the control group of 16 patients, treated with chewing gum containing acetylsalicylic acid and menadione, there was 1 (no. 31) who experienced late bleeding. In none of these 3 patients was the post-tonsillectomy hemorrhage related to a decrease of blood prothrombin, since the prothrombin time on the seventh postoperative day was easily within normal not only in the hemorrhagic patient of the control group but also in the 2 hemorrhagic patients of the group treated with acetylsalicylic acid chewing gum.

SUMMARY AND CONCLUSIONS

The prothrombin concentrations of blood samples taken from 45 cooperative children were estimated by Kato's technic on the day prior to tonsillectomy, on the afternoon following morning operation and one week after operation.

A group of 29 of these patients received chewing gum tablets containing approximately 0.23 Gm. (3½ grains) of acetylsalicylic acid in a dosage of 5 to 6 tablets daily from the first to the seventh postoperative day. A control group of 16 patients received, in the same dosage, chewing gum tablets containing a similar amount of acetylsalicylic acid and, in addition, 0.5 mg. of menadione.

No specific variation of prothrombin time, which normally appears to vary widely, could be ascribed to either acetylsalicylic acid or to acetylsalicylic acid and menadione administered in chewing gum.

Three patients, 2 in the test group and 1 in the control group, experienced late post-tonsillectomy hemorrhage, which bore no etiologic relation to decrease of blood prothrombin.

There is no indication that the routine post-tonsillectomy use of chewing gum containing acetylsalicylic acid may give rise to prothrombinopenic hemorrhage or that such use of this gum has any adverse effect on blood prothrombin.

185 North Wabash Avenue, Chicago.

113 North Thirteenth Street, Newark, N. J.

PLASTIC REPAIR OF THE OBSTRUCTING NASAL SEPTUM

SAMUEL FOMON, M.D.

JOSEPH G. GILBERT, M.D.

A. GILBERT SILVER, M.D.

AND

VICTOR ROYCE SYRACUSE, M.D.

NEW YORK

UP TO 1890 the correction of septal deviations was accomplished by orthopedic redressment—a procedure which contemplates fracturing the septum and maintaining it in the sagittal plane over a period of months by the use of intranasal tubes. The method was successful in the correction of deflections of the bony septum, the friability of osseous structure permitting easy fracture and maintenance in the new position. It was ineffective, however, when the cartilage was involved, for cartilage, because of its great elasticity, is not easily fractured, and the tendency to return to its original form and position remained, despite attempts to overcome this difficulty by cross hatching or removing redundant strips, with or without elevation of the overlying membrane. Further objections were the damage inflicted by the crushing force of the instruments on the membrane, the discomfort of the prolonged postoperative treatment, and the tendency toward atrophy and necrosis of the membranes resulting from the pressure of the tubes.

The generally unsatisfactory results led to the adoption of the septal operation devised by Killian in 1899, and this is still the operation of choice when indicated, despite its many shortcomings, namely: 1. The most ventral (anterior) and caudal (inferior) parts of the septum, which in themselves are frequently the obstructing factors, are not attacked for fear the subsequent contraction of the deskeletonized membranes will result in saddling of the dorsum and distortion of the nasal tip. Inasmuch as the air currents, according to Proetz,[1] pass along these buttresses, it is understandable that the operation must often fail to restore function. 2. The procedure is inapplicable in children. 3. Owing to the extensive removal of cartilage and bone, which in itself violates a surgical principle, a rigid partition is converted into a flaccid membrane which, unable to resist differential intranasal

1. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941.

pressures, has a tendency to sway in the current. With a respiratory pressure in the nose varying from approximately minus 6 mm. of water on inspiration to plus 6 mm. of water on expiration, even the slightest quiver of the membrane, according to Bernoulli's principle,[2] would occasion great physiologic disturbance. 4. Following operation in the case of extreme deviations, the longer membrane remaining on the convex side may buckle and itself create an obstruction. 5. When the deviation involves the ventral buttress, the membranes are prevented from adjusting themselves in the sagittal plane. 6. In the event of laceration, even if slight, there is a tendency toward perforation for the absence of a foundation of supporting structure allows for unhindered retraction of the mucoperichondrial membranes. 7. Finally, if the first operation fails, a second one is mechanically and technically difficult, if not impossible.

Recently a new approach to septal surgery has been evolved, offering greater assurance of functional restoration than could have been expected heretofore. This can be attributed to the following factors:

1. The development of a new concept of the structural anatomy of the septum; i. e., the septum under static conditions does not furnish support to the nasal pyramid but acts only as a reserve safety factor.[3] It follows, then, that saddling of the dorsum and distortion of the nasal tip consequent on a too generous submucous resection result only from tensions developing from the cicatricial pull of the deskeletonized septal membranes—not from a lack of support. Therefore, any part of the septum may be removed without fear of subsequent deformity, provided precautions are taken to neutralize these traction forces.
2. A better understanding of the physiology of the external nose.[1]
3. A more definite placement of the indications by a more careful elimination of extraseptal factors which give rise to symptoms of nasal obstruction, such as allergies, dietary faults and various local factors—for example, deformities of the osseous and cartilaginous pyramids.
4. The application of the technics of plastic surgery.

SURGICAL MANAGEMENT

For surgical purposes we shall divide the nasal septum by an imaginary ventrodorsal (anteroposterior) plane at the weakest part

2. Duff, A. W.: Principles of Physics, ed. 8, Philadelphia, The Blakiston Company, 1946.

3. (*a*) Fomon, S., and others: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141-156 (Aug.) 1946. (*b*) Huffman, W. C.: Personal communication to the author.

of the dorsum, i. e., at the point at which the caudal (inferior) portions of the upper lateral cartilages are attached to the septum (Gilbert). That part lying cephalad to (above) this plane will be called the cephalic compartment and that which lies below will be designated as the caudal compartment (fig. 1). The "weak point" is explainable on the following grounds: The anatomic arrangement whereby the upper lateral cartilages are attached to the under surfaces of the nasal bones and the frontal processes of the maxillas is such as to furnish sufficient stability to maintain the projection of the profile under static conditions. Obviously, the weakness of the cartilaginous dorsum is in direct proportion to the distance it extends from its bony attachment. The weakest part of the dorsum, therefore, must be the area just caudal to the point at which the upper lateral cartilages are attached

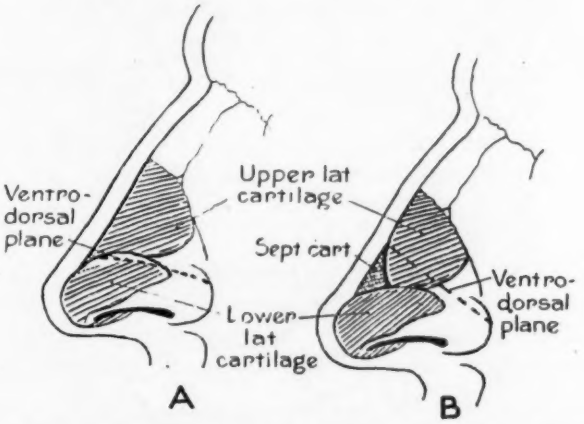


Fig. 1.—Division of the septum by a ventrodorsal plane passing through the point at which the upper lateral cartilages are attached caudally to the septum, indicated by the dotted line. *A*, upper lateral cartilages extending to the caudal end of the septum: Complete removal of septum will not cause saddling. *B*, upper lateral cartilages swinging away from the septum above its caudal margin: Submucous resection likely to result in saddling.

to the septum. This "weak point" is buttressed by the overlapping lower lateral cartilages. Thus, in those instances in which the upper lateral cartilages swing away from the septum at an unusually high level (cephalad) (fig. 1 *B*) and/or in which the lower lateral cartilages are underdeveloped or soft in texture, even minimum trauma may result in saddling. In cases in which this anatomic weakness is found to exist, it is essential that every precaution be taken at operation to neutralize the effect of postoperative traction forces at this site. This is best accomplished by implanting two or more battens, one in the columella and the other or others above (cephalad) between the freed septal membranes. The deskeletonized membranous septum lying

between the battens becomes a zone of dispersion for the contracting forces. If but one batten were employed, buckling of the cartilage, saddling of the dorsum and retraction of the columella might result from the uninterrupted traction forces (fig. 2). When the upper lateral cartilages swing away from the septum at an unusually high (cephalad) level and the lower lateral cartilages are not resilient, it may be advisable to supplement these battens by another implanted subcutaneously along the long axis of the dorsum as an additional safety factor.

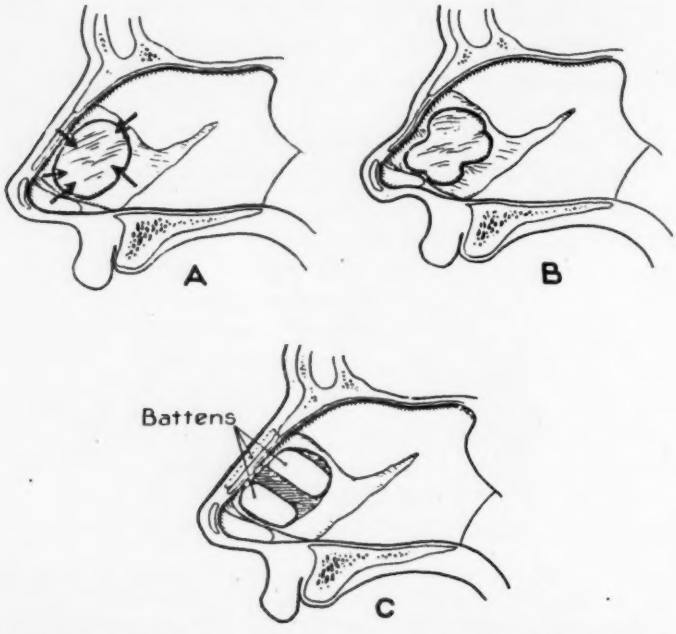


Fig. 2.—Effect of single vs. multiple battens. *A*, traction forces following submucous resection. *B*, sequelae of such forces. *C*, battens in situ counteracting traction forces.

When the supporting elements at the "weak point" are adequate—i. e., the upper lateral cartilages extend to the end of the septum, and the lower lateral ones are well developed and resilient (fig. 1 *A*)—no batten is necessary. Nevertheless, strips of the resected cartilage are placed between the septal membranes, not to counteract tension, but merely to restore the rigidity of the septum.

SEPTAL OBSTRUCTION NOT ASSOCIATED WITH EXTERNAL NASAL DEFORMITY

Deflections Limited to the Cephalic Compartment.—For all deviations of the cephalic compartment the orthodox Killian operation is satisfactory, since the tensions developing in the deskeletonized mem-

brane operate from fixed bony points, and the construction of the pyramid is so sturdy that no amount of contraction is likely to cause saddling of the dorsum. After removal of the bone and the cartilage, the nasal fossae are packed, and the cartilage battens are introduced between the septal flaps and held in place by the pressure of the nasal packing.[4] Should the resected bone or cartilage not be usable, preserved cartilage is employed. The purpose of these battens is not to neutralize tensions but to restore the rigidity of the septum.

Deflections Limited to the Caudal Compartment.—In deviations involving the caudal end of the septum, our experience with the Metzenbaum [5] operation, commonly employed for deformities in this location, has been disappointing due to the tendency of the septum to swing back to its original position. The factors contributing to this tendency are the inability of the membranes to readapt themselves to the new position, owing to their elevation on one side only, and the failure to take into account the associated deformity of the upper lateral cartilages, which, acting as guy ropes, exert unequal traction on the septum. Other objections are that the procedure fails to straighten the buckled cartilage despite latticing and strip excisions. And should the deformed cartilage be thickened and comminuted, even when brought into the midline it still remains a source of obstruction.

These faults paved the way to the introduction of the Galloway operation,[3a] and while this operation is a great improvement as compared with that of Metzenbaum, its application is strictly limited to the most caudal displacements of the septum. In the correction of more extensive dislocations it is inapplicable in that the use of a single batten fails to break the line of force, culminating frequently in retraction of the columella and buckling of the implanted cartilage (fig. 2 *B*).

Technic of the Galloway Operation: The membranous septum is incised on the right side throughout its extent with a no. 15 Bard-Parker knife, perforation of the opposite side being guarded against by the index finger held in the left vestibule (fig. 3*A*). A bed is created in the columella by separating the two laminas with a no. 15 Bard-Parker knife, supplemented with a Stevens scissors (fig. 3*B*). Two silk sutures are passed through the mucoperichondrium, and the membrane is elevated on one side (fig. 3*C*). The mucoperichondrium on the opposite side is elevated in a like manner (fig. 3*D*). The septal cartilage is removed with scissors or a swivel knife (fig. 3*E*).

4. Cottle, M. H.: Personal communication to the author.

5. Metzenbaum, M. F.: Recent Fracture of Nasal Base Lines of Both Outer Nasal Walls, with Divergent Displacement: Orthopedic Procedures for Obtaining Anatomic Reduction of Osseous and Cartilaginous Nasal Framework, Arch. Otolaryng. **34**:723-735 (Oct.) 1941.

To overcome the subsequent tensions arising from the contraction of the deskeletonized membrane, either the resected septal cartilage, if usable, or preserved cartilage is reinserted. The size of the cartilage should be such as to extend not farther than the "weak point"—i.e., the point at which the caudal (inferior) ends of the upper lateral cartilages are attached to the septum. A larger piece would have a tendency to cause buckling and retraction of the columella. To restore the rigidity of the membrane, smaller pieces are implanted and held in place by the nasal packing. Two pilot sutures are passed through that part of

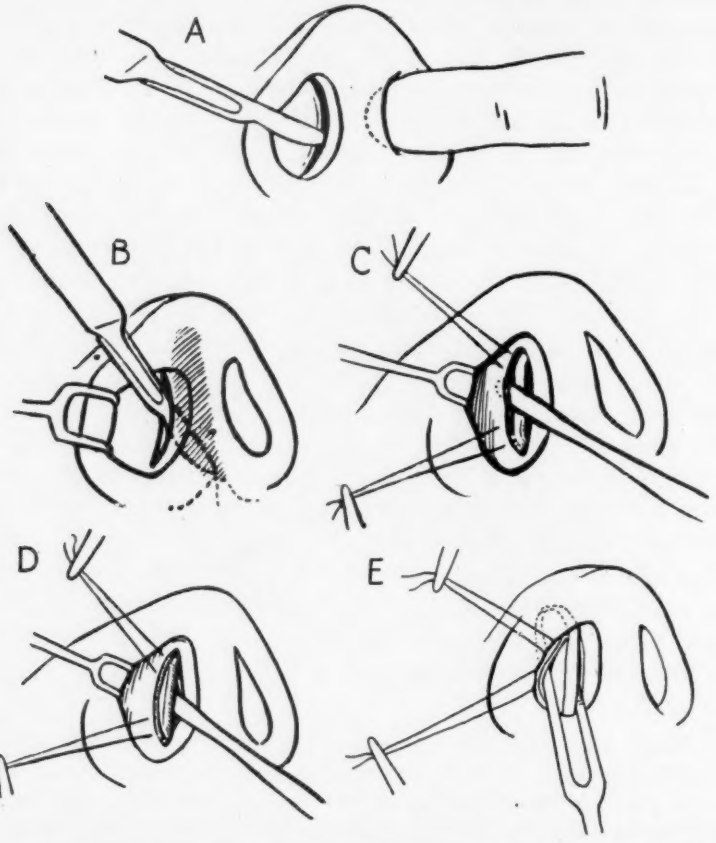


Fig. 3.—Galloway operation: *A*, membranous septum incised, the index finger protecting the opposite wall against perforation.

B, bed prepared in the columella for reception of the graft.

C, mucoperichondrial flap held aside with two mattress sutures and elevated.

D, mucoperichondrial flap on the opposite side elevated.

E, obstructing septum removed with swivel knife.

the cartilage which is to fit into the columellar bed. The cartilage graft is then introduced between the membranes until lost from view (fig. 4*A*). The suture ends emerging from the cartilage are passed through two straight needles, which are introduced into the previously prepared bed, and brought out to the surface at the center of the columella (fig. 4*B*). Traction is exerted by the assistant to

draw the cartilage into the columellar bed (fig. 4*C*), where it is fixed by two mattress sutures passing through the full thickness of columella and graft (fig. 4*D*). The pilot sutures, having subserved their purpose, are withdrawn.

For more extensive displacements we have abandoned the use of the Galloway operation in favor of the following one:

Technic of the Fomon Operation: The membranous septum is incised on the right side throughout its extent with a no. 15 Bard-Parker knife, perforation of the opposite side being guarded against by the index finger held in the left vestibule (fig. 3*A*). A bed is created in the columella by separation of the two

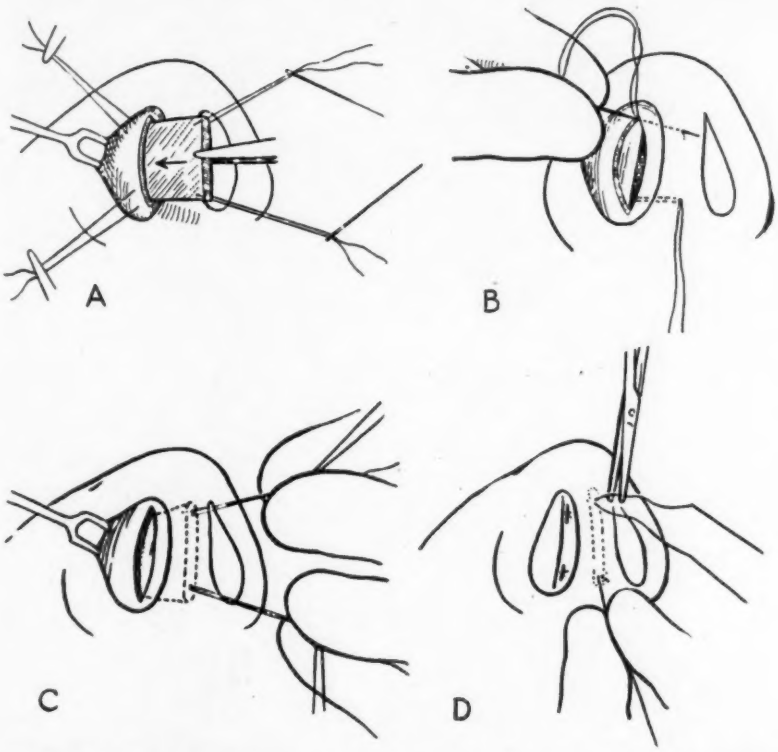


Fig. 4.—Galloway operation continued: *A*, cartilage introduced between the mucoperichondrial flaps. Two sutures are passed through the cartilage to pilot the graft into the columellar bed.

B, needles introduced into the columellar bed and brought out through the columellar skin.

C, traction exerted on sutures to draw the cartilage into the columellar bed.

D, cartilage fixed into the columellar bed with two mattress sutures. The pilot sutures are removed.

laminas with a no. 15 Bard-Parker knife, supplemented with a Stevens scissors (fig. 3*B*). The two layers are held apart with dural hooks, and a cartilage graft is introduced (fig. 5*A*) and fixed in place by one or two through and through silk mattress sutures passed behind the graft (fig. 5*B*). The width and thick-

ness of the graft will vary from 2 to 4 mm., depending on the amount of contraction anticipated. Its length will average 2.5 to 3 cm., depending on the projection of the tip.

It is to be emphasized that this cartilage graft is not introduced as a support, since it presents no abutment to the lower lateral cartilages ventrally or to the nasal spine of the maxillas dorsally. Its purpose is merely to act as a batten and prevent the retraction of the columella occasioned by the subsequent inevitable pull of the contracting connective tissue of the septal flaps.

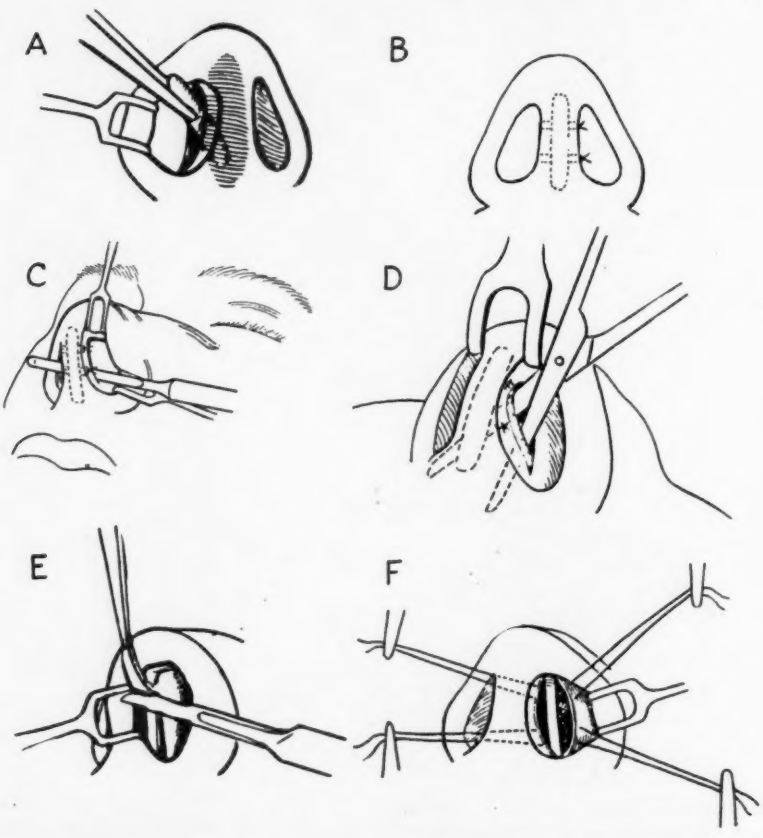


Fig. 5.—Fomon technic: *A*, columellar graft inserted into the prepared pocket.

B, graft held in place by sutures passed behind it.

C, membranous septum cut through to complete transfixion.

D, caudal exposure completed. The membranous septum is straddled by Mayo scissors, and the structures are divided to the nasal spine.

E, sliver resected from the caudal end of the septum to delineate the line of cleavage.

F, suture threads passed through mucoperichondrium serving as automatic retractors.

With a no. 11 Bard-Parker knife, the membrane on the opposite side is cut through, and the transfixion is thus completed (fig. 5 *C*). With a straight Mayo

scissors held perpendicular to the nasal spine, the membranous septum is straddled, and with one snip of the scissors it is completely divided down to the nasal spine (fig. 5*D*).

By retracting the lower lateral cartilaginous vault to one or the other side, the septum is brought into clear view. With a Bard-Parker no. 11 knife, a thin sliver is removed from its caudal margin to furnish a clearer delineation of the line of cleavage (fig. 5*E*). A curved needle armed with heavy silk thread is passed through the caudal margin of the mucoperichondrium on the left side. The suture ends are grasped with a hemostat, which is thrown over the side of the face to act as an automatic retractor. A similar suture is passed at a more ventral level. The caudal margin of the mucoperichondrium on the right side is similarly treated

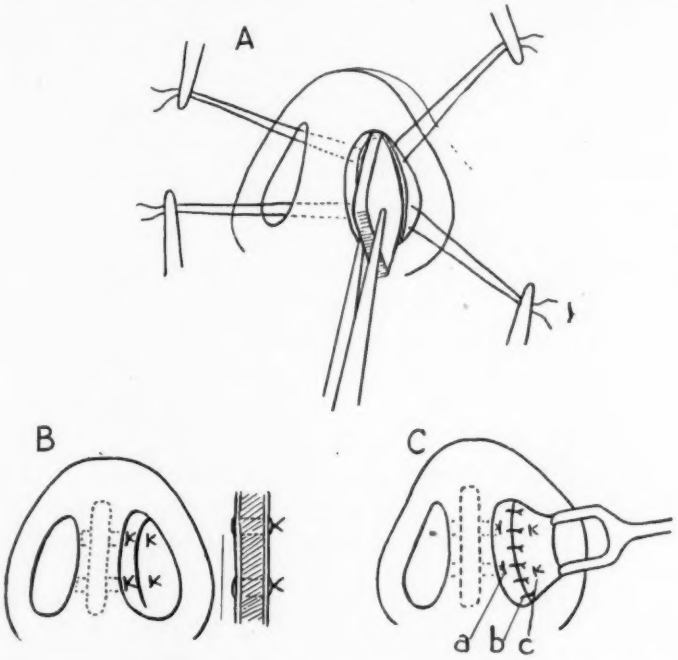


Fig. 6.—Fomon technic continued: *A*, cartilage inserted and covered by the mucoperichondrial flaps.

B, cartilage secured in place by mattress sutures.

C, raw surfaces approximated: (*a*) mattress sutures holding columellar graft in place; (*b*) atraumatic sutures uniting the mucoperichondrial flaps to the columellar skin; (*c*) sutures holding septal grafts to mucoperichondrial flaps.

(fig. 5*F*). Elevation of the membrane is accomplished in the usual manner, the extent depending on the deformity.

The obstructing cartilage, irrespective of its location, is resected with scissors or a swivel knife as far ventrad or caudad as is necessary (fig. 3*E*). Inasmuch as the septum is a redundant member under static conditions, one need not fear immediate saddling of the dorsum or dropping of the tip except in cases such as that shown in figure 1 *B*. Subsequent contractile deformities are prevented by the reinsertion of cartilage, the choice of its source depending on conditions present. If the resected cartilage is usable or can be made usable by shaving or cross hatching,

it is preferable. Otherwise, isografts of septal cartilage preserved in merthiolate solution [6] are employed.

If the cartilage is a large single piece, it is held with a rat tooth forceps by the assistant, and the two flaps of mucoperichondrium are brought to the midline on each side of the cartilage (fig. 6*A*). Two mattress sutures of silk thread are passed through both the flaps of mucoperichondrium and the septal cartilage (fig. 6*B*). The mucoperichondrial flaps on each side are reunited to the skin of the columella with no. 5-O atraumatic dermal sutures to eliminate all exposed raw surfaces (fig. 6*C*). If the septal cartilage came away in several small pieces, the nasal fossae are first packed, and then the cartilage is introduced between the membranes, being retained in place by the pressure of the packing.

The nasal fossae are packed with petrolatum gauze under sufficient pressure to apply the membrane to the cartilage graft. The collection of secretions and the development of hematomas are prevented by the absence of dead spaces and the dependent drainage. The sutures are removed on the fourth or the fifth day.

Deflections Occurring in Both the Cephalic and the Caudal Compartment.—Deviations of the perpendicular plate and the upper part of the quadrilateral plate are corrected by the Killian procedure. The caudal deformity is rectified by the procedure described in the foregoing sections.

SEPTAL OBSTRUCTION ASSOCIATED WITH EXTERNAL NASAL DEFORMITY

Satisfactory results will depend as much on the sequence of the operative steps as on the care with which each step is carried out. Irrespective of the location of the obstruction: 1. The soft structures over the nasal pyramid are elevated. A unilateral incision is made along the entire extent of the membranous septum, through which a pocket is made in the columella. Into this a batten of cartilage is introduced and the pocket is closed with two mattress sutures. The other surface of the membranous septum is then cut through. 2. The projection of the profile is reduced. If the septal procedure were to precede this step, there would be a likelihood of the mucous membranes' being caught in the saw and torn from their attachments. 3. The septal obstruction is corrected. 4. The upper lateral cartilages are cut from the septum. The reason for postponing this step until now is that one is enabled to take advantage of the stabilizing action of the upper lateral cartilages on the septum while it is being resected. 5. The nose is narrowed. This step is best done at this time, for the septal hindrance has been removed. 6. The lobule is modeled. 7. Finally, the cartilage is replaced as a batten between the septal membranes.

Cephalic Compartment.—Deflections of the perpendicular plate, even if minor and occasioning no physiologic disturbance, must be corrected, and the Killian operation is the one of choice (fig. 7). If not corrected, the deflection may interfere with narrowing of the nose, prevent sym-

6. Fomon, S.: Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Co., 1939.

metric alinement of the bones and incur the possibility of postoperative obstruction of the surgically narrowed nose.

Caudal Compartment.—Here the choice of procedure will depend on the character of the deformity of the most ventral (anterior) part of the septum.

1. When the ventral margin of the septum is straight and in the midline—in other words, when it is not a party to the obstruction—no problem is offered. The rhinoplasty is performed in the customary

Fig. 7.—Reconstruction of a septum to permit of narrowing and also to prevent postoperative obstruction following rhinoplasty. (Courtesy of Dr. Markey Pullen.)

manner, and the dorsocaudal (posteroinferior) septal correction is carried out according to the Fomon technic outlined in a foregoing section of this paper. In the correction of such deformities the upper lateral cartilages may or may not be cut away from the septum, depending on the rhinoplastic indications.

Fig. 8.—Reconstruction of a septum as described on page 19 under "Caudal Compartment" second procedure. (Courtesy of Dr. Edmonde Neer.)

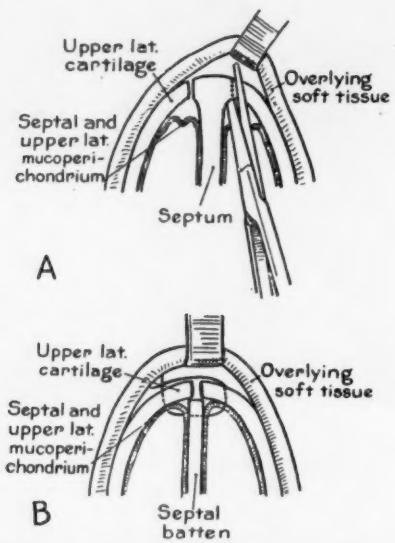


Fig. 9.—*A*, mucoperichondrium separated from the septum and the upper lateral cartilages. The overlying soft tissues are elevated. The upper lateral cartilages are cut from the septum.

B, batten between the septal membranes fixed to the upper lateral cartilages by catgut sutures.

2. When the ventral margin is straight but deviated from its central position, the procedure just described is employed, and, in addition, the upper lateral cartilages, which here are always a party to the deformity, are severed from the septum and the longer one (on the side opposite the deflection) is reduced to equal the size of the shorter one (fig. 8). Otherwise, the upper lateral cartilages, which serve as guy ropes for the septum, will prevent its being shifted into the midline.

3. In C-shaped or S-shaped deformities of the ventral margin, the upper lateral cartilages are always involved and require trimming, straightening, cross hatching and/or resection. Here, in order that the septal membranes may remain suspended and not be lost in the nose, the upper lateral cartilages are cut from the septum submucously

Fig. 10.—S-shaped deformity of the septum and upper lateral cartilages corrected as described on this page under "Caudal Compartment," third procedure. (Courtesy of Dr. Louis Kleinfeld.)

as follows (fig. 9*A*): The septal membranes are elevated in the customary manner and the dissection continued beneath the upper lateral cartilages. An angulated retractor is introduced to guard the overlying skin, and the upper lateral cartilages are cut away from the septum with a no. 11 Bard-Parker knife or scissors, the mucosa being left intact. The septum is now removed, after which the upper lateral cartilages are treated as indicated. A batten of cartilage is introduced between the septal membranes and fixed to the upper lateral cartilages by one or two catgut sutures (fig. 9*B*; fig. 10).

Cephalic and Caudal Compartments.—Deviations of the perpendicular plate and the upper part of the quadrilateral plate are corrected by the Killian procedure. The caudal deformity is rectified by one of the three technics just described under "Caudal Compartment."

COMMENT

Generally speaking, a better result will be obtained if the rhinoplasty and the septal correction are done in separate operative stages three or more weeks apart. Inasmuch as the final outcome of the operation depends in a measure on the healing qualities of the tissues, the smaller amount of trauma inflicted by dividing the stages speaks for less reparative reaction and a better anatomic and physiologic result.

As to the order of sequence, the old teaching advocated that the rhinoplasty precede the submucous resection on the grounds that the septum supported the nose and that if the submucous resection were done first a subsequent removal of a hump might open into the resected area and result in saddling. New concepts of the structural anatomy of the septum have proved this assumption fallacious, and today the submucous resection is done first since it permits an easier osteotomy and a more symmetric alinement of the pyramid. But there are occasions when it is impossible to restore breathing without simultaneously correcting the external deformity and the deviated septum. For instance, if there is a long nose with a caudal displacement of the septum, resection of the lower part of the septum to shorten the nose automatically eliminates the deflection. And in cases in which the deviated septum is a part of an S-shaped or deflected nasal pyramid, it would be impossible to place the septum in the sagittal plane without an accompanying rhinoplasty.

CHEMICAL MENINGITIS FOLLOWING USE OF TYROTHRICIN

A Clinical and Experimental Study

FRANK J. OTENASEK, M.D.
AND
DAVID FAIRMAN, M.D.
BALTIMORE

TYROTHRICIN, a bactericidal substance, has been known since 1939, when it was first discovered by Dubos,[1] who the following year [2] elaborated on its effect in experimental bacterial infections. His description was rapidly followed by numerous clinical trials, which have been enthusiastically recorded in the medical literature from 1941 to the present time. Its effectiveness when it is used locally in the treatment of infections of the nasal sinuses and the mastoid process has been repeatedly pointed out by Crowe and his associates.[3] The toxic and the pharmacologic properties of the new agent were early studied by Herrell and Heilman,[4] Rammelkamp and Weinstein [5] and Robinson and his associates.[6] In general, the material was shown to

From the Department of Surgery (Neurosurgery), Johns Hopkins University and Hospital.

1. Dubos, R. J.: Studies on a Bactericidal Agent Extracted from a Soil Bacillus, J. Exper. Med. **70**:1 (July) 1939.

2. Dubos, R. J.: The Effect of Specific Agents Extracted from Soil Micro-Organisms upon Experimental Bacterial Infections, Ann. Int. Med. **13**:2025 (May) 1940.

3. Bordley, J. E.; Crowe, S. J.; Dolowitz, D. A., and Pickrell, K. L.: The Local Use of Sulfonamides, Gramicidin (Tyrothricin) and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **51**:936 (Dec.) 1942. Crowe, S. J.; Fisher, A. M.; Ward, A. T., Jr., and Foley, M. K.: Penicillin and Tyrothricin in Otolaryngology, ibid. **52**:541 (Sept.) 1943. Crowe, S. J., and Ward, A. T., Jr.: Local Use of Sulfadiazine, Tyrothricin, Penicillin and Radon in Otolaryngology, North Carolina M. J. **4**:431 (Oct.) 1943. Crowe, S. J.: The Local Use of Sulfadiazine Solution, Radon, Tyrothricin and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **53**:227 (June) 1944.

4. Herrell, W. E., and Heilman, D.: Experimental and Clinical Studies on Gramicidin, J. Clin. Investigation **20**:583 (Sept.) 1941.

5. Rammelkamp, C. H., and Weinstein, L.: Hemolytic Effect of Tyrothricin, Proc. Soc. Exper. Biol. & Med. **48**:211 (Oct.) 1941; Toxic Effects of Tyrothricin, Gramicidin and Tyrocidine, J. Infect. Dis. **71**:166 (Sept.) 1942.

6. (*a*) Robinson, H. J., and Molitor, H.: Some Toxicological and Pharmacological Properties of Gramicidin, Tyrothricin and Tyrocidine, J. Pharmacol. & Exper. Therap. **74**:75 (Jan.) 1942. (*b*) Robinson, H. J., and Graessle, O. E.: In Vitro and in Vivo Studies of Gramicidin, Tyrothricin and Tyrocidine, ibid. **76**: 316 (Dec.) 1942.

be markedly toxic (mostly by hemolysis) when used intravenously or by other injection method. In animal experiments its use was limited to surface application, on infected areas, or to irrigation of infected body cavities. It was shown that tyrothricin could not be used indiscriminately in the larger body cavities, but only in amounts small enough to have a local bacteriostatic effect without producing general effects.[6a]

We have been able to find no published accounts of ill effects occurring in human beings from clinical use of tyrothricin and therefore deem it important to make known 2 cases in which patients seen in the Johns Hopkins Hospital suffered disastrous effects from the application of this drug.

REPORT OF CASES

CASE 1.—J. E., a 35 year old white man, was admitted to the otolaryngolic service of the Johns Hopkins Hospital, Jan. 28, 1943, because of postnasal drip and right frontal headache. Examination showed only exquisite tenderness over the right frontal sinus. There was clouding in the roentgenograms. A diagnosis of an acute flare-up of chronic right frontal sinusitis was made, and drainage of the sinus was instituted at once through a small trephine opening. Four days later a somewhat more radical operation was carried out. Cultures had grown Staphylococcus aureus. Postoperatively the sinus was frequently irrigated both from above and through the normal nasal opening with a solution of tyrothricin (1:10,000). The patient's wound healed well, and he was discharged from the hospital in three weeks (February 17). Two days later another irrigation was carried out in the nose and throat outpatient department. The patient fainted immediately after the irrigation, and when aroused he was confused, complained of headache and vomited. He was readmitted to the hospital, where, within a few hours, nuchal rigidity developed and his temperature rose to 103 F. The white blood cell count was 28,000. Lumbar puncture revealed cloudy spinal fluid containing 15,000 white cells, all polymorphonuclears. On the supposition that the patient had bacterial meningitis, penicillin and sulfadiazine therapy was instituted. However, repeated cultures of the cerebrospinal fluid were sterile. After three days the spinal fluid cell count had come down to 400, of which 50 per cent were mononuclear. The patient was semicomatose for nearly one week and had several generalized convulsions, but gradually improved. His temperature was normal after two weeks, and the spinal fluid was acellular after that time. The patient was discharged as well on March 20, a little more than a month after the onset of meningitis.

He was readmitted to the medical service of the hospital on April 9, 1943, because of persistent occipital headache, vomiting, drowsiness and confusion. There was slight and inconstant bradycardia, also weakness of the left side of the face of the upper motor neuron type. The spinal fluid pressure was 340 mm. of water; the fluid contained no cells. It was felt that the patient had a cerebral abscess of the right side, and ventriculography was carried out. The roentgenograms showed hydrocephalus, with symmetric dilatation of all the ventricles and no air in the subarachnoid spaces. A cerebellar exploration revealed adhesive arachnoiditis occluding the basal cisternae. Choroid plexectomy of the fourth ventricle was done, as it was clear that a communicating hydrocephalus was present. Convalescence was rather stormy, but the patient eventually left the hospital with a cerebrospinal fluid pressure of 180 mm. of water. His suboccipital decompression was soft. There was no perception of light in the right eye, and there was marked

disturbance of visual acuity in the left. Except for this visual defect and some unsteadiness, the patient remains normal to this date.

CASE 2.—T. K., a 32 year old white man, was admitted to the otolaryngologic service of the Johns Hopkins Hospital, March 8, 1945, because of a discharging sinus above the right eye. This had followed an automobile accident ten years before and was shown to be due to chronic infection and osteomyelitis of the frontal sinus. There was also infection of the ethmoid cells. Ethmoidectomy was done, followed on March 13, by radical right frontal sinusotomy. A Dakin tube

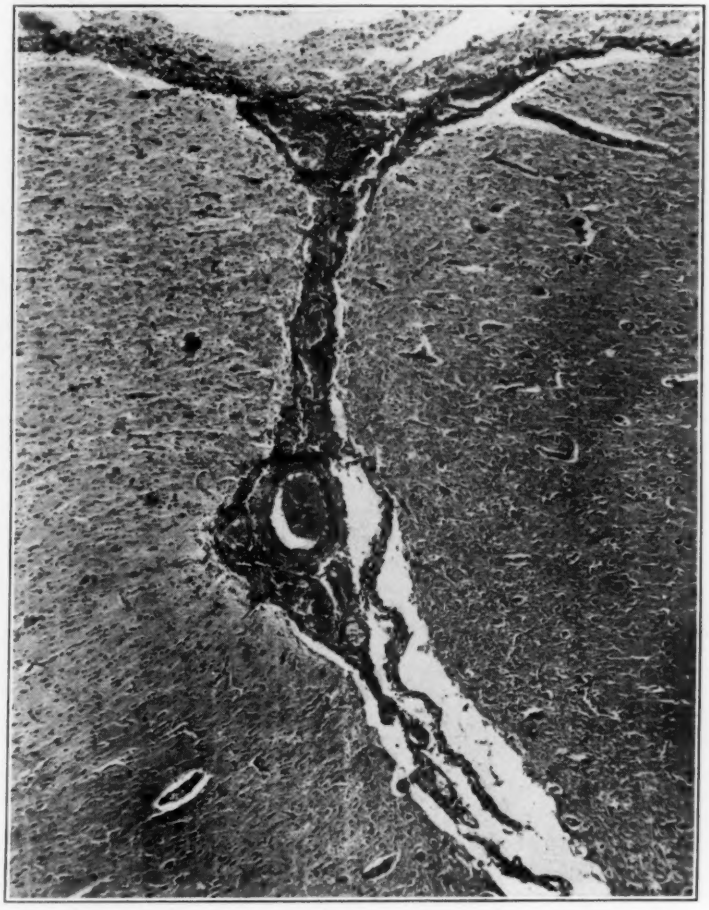


Fig. 1.—Autopsy specimen of the leptomeninges, showing thickening and round cell infiltration (case 2); × 60.

was left in place for subsequent irrigation. Two days later, during the course of an irrigation with a solution of tyrothricin (1 : 10,000), the patient experienced a sudden stabbing occipital headache and became cold and clammy. In a short time he became delirious, and within six hours his temperature rose to 104.4 F. Lumbar puncture was performed, and the spinal fluid showed 2,000 cells per cubic millimeter, more than 70 per cent of which were polymorphonuclears. Cultures were sterile. The patient remained quite ill for one week, after which his temperature

returned to normal and he seemed well. He was discharged March 26, eleven days after the abrupt onset of his meningitis.

About two weeks later, on April 11, the patient was readmitted because of persistent headache, vomiting and some confusion. There was marked bradycardia but no localizing sign of an intracranial lesion. The optic disks showed early blurring. Ventriculography was carried out. There was tremendous pressure, and the ventricles were large, 150 cc. of fluid being obtained. The roentgen films again showed symmetric dilatation of the ventricular system, with no air in the subarachnoid space. An attempted ventriculostomy was carried out, but the patient succumbed on April 26, of uncontrolled intracranial pressure.

Postmortem examination revealed hydrocephalus and chronic meningitis with thickening and round cell infiltration of the leptomeninges (fig. 1). There

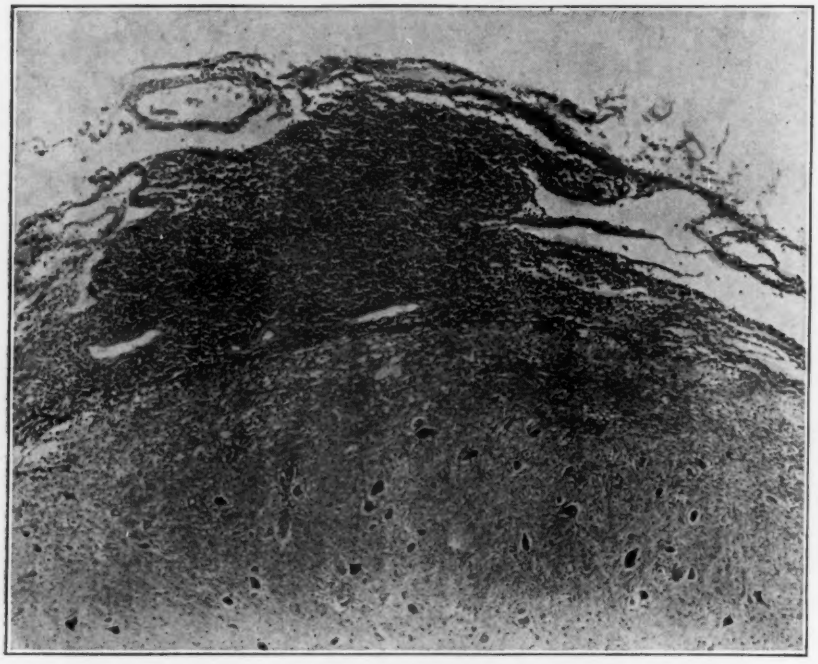


Fig. 2.—Acute purulent exudate.

was a break in the dura behind the posterior plate of the frontal sinus which permitted the irrigating fluid to flow into the subarachnoid space.

EXPERIMENTAL STUDIES

These 2 cases had much in common. In both, morbid effects were seen immediately after a frontal sinus had been irrigated with tyrothricin. They were severe headache, delirium, high fever and, in a very few hours, clinical and laboratory signs of meningitis. In both, the acute inflammatory process lasted less than a week, with apparent clinical cure but with delayed production of hydrocephalus as a consequence of adhesive arachnoiditis.

These two clinical experiences prompted a limited laboratory investigation of the pathologic processes involved. Thirty-seven dogs were used in the experiments. At first the method employed for the administration of tyrothricin was to make a trephine opening in the vertex of the skull, incise a small opening in the dura and close the wound, with a catheter being left in the extradural space for subsequent irrigation. This method proved unsatisfactory, for even with the dura

Fig. 3.—Exudate showing predominantly polymorphonuclear cells; × 300.

open the irrigating fluid did not always reach the subarachnoid space. We therefore injected 1 cc. of a suspension of tyrothricin (1:1,000) directly into the cisterna magna. The stock solution (2 per cent in 94 per cent alcohol) was used, suitably diluted. Thirty-five animals received such injections. Of these, 18 died either immediately or within a few hours. Those with a survival period of a few hours showed massive swelling of the medulla oblongata at the site of injection, with almost complete obliteration of the foramen of Magendie in some cases.

Further studies were carried out on the 17 animals which had survived longer periods. In all there was pleocytosis of the spinal fluid, the white cell counts being in the neighborhood of 2,000 to 5,000 during the first forty-eight hours after injection. The white cell counts returned to normal after ten days. In many instances cell counts of the spinal fluid were impossible to make, for clotting of the fluid occurred immediately after withdrawal. In these the protein determinations were as high as 950 mg. per hundred cubic centimeters. Bacterial cultures were uniformly negative.

The surviving animals were killed at periods ranging from twenty-four hours to four months after a single injection. In general, the

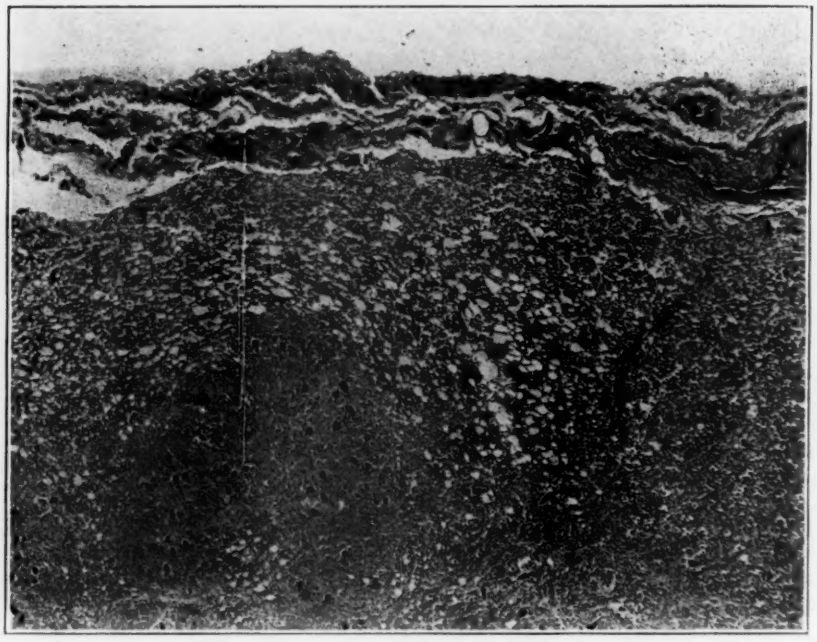


Fig. 4.—Exudate in which the polymorphonuclears have been succeeded by mononuclear cells. There is also laying down of fibrin; × 59.4.

pathologic findings in the first twenty-four to forty-eight hours were consistent with acute purulent meningitis, the exudate consisting largely of fibrin and polymorphonuclear leukocytes (figs. 2 and 3). After several days the picture changed to that of a soft organizing exudate with a preponderance of mononuclear cells (fig. 4). At this stage, also, encroachment on the foramen of Magendie was often noted.

The final picture revealed gross and microscopic thickening of the leptomeninges, with or without hydrocephalus (fig. 5). Only one animal, which was killed nearly four months after the initial injection, was observed to have full-blown hydrocephalus involving all the ventricles (fig. 6).

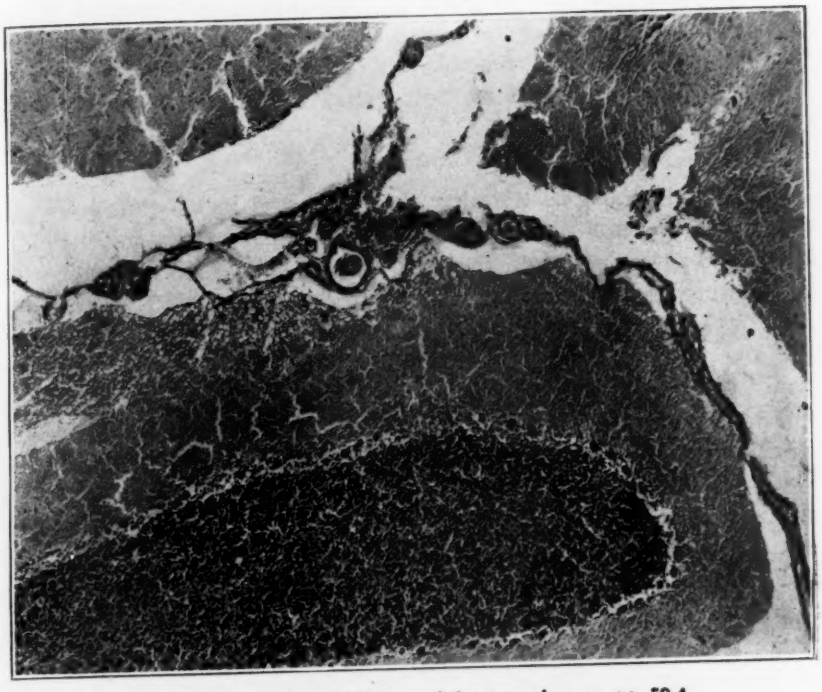


Fig. 5.—Chronic thickening of leptomeninges; × 59.4.

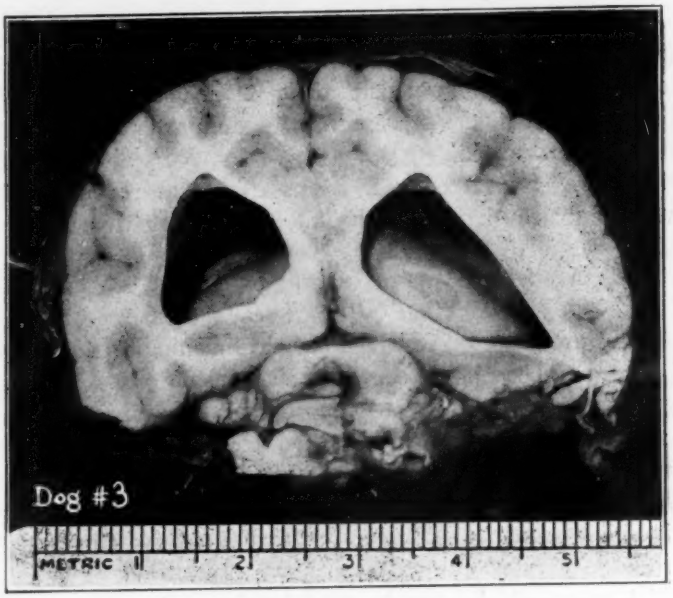


Fig. 6.—Hydrocephalus.

Inasmuch as the stock solution of tyrothricin is suspended in 94 per cent alcohol, 2 dogs were each given an injection of 1 cc. of a 1:10,000 solution of alcohol as a control. In one of these, pleocytosis of the spinal fluid (only 50 mononuclear cells) occurred; in the other no changes were noted.

COMMENT

These investigations traced in 2 patients and in laboratory animals the course of events which follows clinical instillation of a suspension of tyrothricin if this gets into the subarachnoid space. In one patient an opening in the dura could be demonstrated at postmortem examination; in the other such an opening must be presumed to have been present. We were able to follow the patients through the various phases of meningitis resulting from chemical irritation. This began with the appearance of an acute purulent exudate not distinguishable from that of acute bacterial meningitis except by reason of negative cultures. The patients were followed during a transitory "subacute" stage of meningitis, in which there was a preponderance of mononuclear cells in the spinal fluid. This stage was followed in both by apparent cure and relief of symptoms, only to go over into a third stage in which there was increased intracranial pressure without cellular evidence of meningeal irritation. This is shown to be due to chronic thickening of meninges with occlusion of the basal cisternae, resulting in a communicating type of hydrocephalus.

These observations serve to illustrate, first of all, that even very small amounts of tyrothricin in the cerebrospinal fluid may produce disastrous effects. Secondly, they indicate that when an opening of the dura mater is known to exist after a radical sinusotomy, tyrothricin irrigations should be scrupulously avoided. Thirdly, they imply that a small opening is sometimes present in the dura after a sinus operation, even though unsuspected.

Under these circumstances it seems clear that routine and indiscriminate tyrothricin irrigation of infected cavities close to the subarachnoid space is to be avoided.

SUMMARY

Two cases are reported in which chemical meningitis developed after a nasal sinus had been irrigated with a suspension of tyrothricin. The pathologic process was recreated in animal experiments, which are described. The profound changes noted are sufficient to raise a note of caution as to whether suspensions of tyrothricin should continue to be used for irrigating sinus cavities that are in close proximity to the subarachnoid spaces. It is hoped that the observations here made will encourage greater efforts toward the production of a less toxic bactericidal agent which may be used locally under these conditions.

6 East Eager Street.

NONVIBRATORY TINNITUS

Factors Underlying Subaudible and Audible Irritations

EDMUND PRINCE FOWLER, M.D.
NEW YORK

FOUR YEARS AGO, before the American Laryngological, Rhinological and Otological Society, Inc., I read a paper entitled "The Control of Head Noises." [1] The opening paragraph in that paper seems appropriate for repetition here. It stated:

> If one disregards the auditory hallucinations occurring in organic diseases of the brain and in the psychoses, there are two kinds of tinnitus: (1) vibratory, caused by actual autogenous vibrations reaching the ear from any part of the body, and (2) nonvibratory, caused by biochemical irritation of the auditory neural mechanism.[2] Either of these two kinds of tinnitus may be superimposed on the other, in which case either one with sufficient intensity and proper frequency can diminish or increase the loudness and change the timber and therefore the degree of annoyance of the other.

In the present paper I shall deal with the second or nonvibratory type of tinnitus and shall discuss means for exploring it from the standpoint of etiology and methods for its prevention, differential diagnosis and control.

Nonvibratory tinnitus is caused by an irritation of the auditory nerve or, more properly, the auditory neural elements. It is an illusion of sensation. If it is misinterpreted, it becomes a delusion. As a matter of fact, I believe it is seldom misinterpreted. It is disturbing more because of its persistency and because of an exaggerated idea of its intensity than because of its actual intensity. It is most annoying when associated with psychologic and somatic disturbances of the ear or of other parts of the body. Tinnitus is a common symptom of aural disorders. In my own practice it is encountered in people past middle age in over 85 per cent of the cases, and in old people even more frequently. Children seldom complain of it. When they do, it is apparently very intense or associated with impending spells of deafness or vertigo.

---

1. Fowler, E. P.: Control of Head Noises: Their Illusions of Loudness and of Timbre, Arch. Otolaryng. **37**:391 (March) 1943.

2. Fowler, E. P.: (*a*) Head Noises and Deafness: Peripheral and Central, Laryngoscope **49**:1011 (Oct.) 1939; (*b*) Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

In order to render the greatest service to those who suffer from tinnitus, one should be familiar with the fundamentals of sensory nerve physiology and with neural reactions to pathologic stimulations. As far as known, the auditory nerve functions, both normally and pathologically, in the same way as other sensory nerves. Therefore, it may respond to irritations applied anywhere along its course—in other words, at any level from the peripheral end organ to the cerebral cortex. Since there is a frequency distribution of the neurons at each level, a discrete as well as a band frequency tinnitus may have its point of origin directly or indirectly in one or both auditory end organs and at any or all levels, or on one or on both sides of the brain. It is therefore certain that tinnitus, like the threshold audiogram, represents a combination or a summation of whatever lesions affect the peripheral and the central auditory mechanisms.

One knows superficially of many things which may cause tinnitus. Common exciting agents are: pressure of impacted cerumen; reduction of the patency of the eustachian tube; inflation, deflation or inflammation of the middle ear and adjacent spaces. The neural and vascular end results of psychologic upsets are legion. The ear has a sensitive neural mechanism with a terminal blood supply. It is perhaps more sensitive than any other organ affected by neurovascular imbalance. In sufficient dosage for the patient involved, alcohol, ether, quinine, the salicylates, streptomycin, caffeine and various other stimulants and depressants, as well as poisoning by gases, foods, drugs and beverages, may set up tinnitus. Infections, simple or pernicious anemia, cardiovascular disease or disturbance of the circulation of the blood or the lymph in the head due to edema or inflammation or to applied means, or to contraction of the muscles about and within the head and the neck may all cause tinnitus. Excessive smoking, auditory fatigue, acoustic or other trauma, severe changes of barometric pressure in the air, on the earth or under the sea, and even muscular exhaustion, overwork, fright and emotional shocks without any trauma from without are conspicuous common causes. In some people allergy or the presence in the blood of some foreign protein plays a major part in the causation, and in numerous instances tinnitus is associated with vasomotor rhinitis. Digestive disorders, whether due to a fault of absorption or to one of elimination, seem to be too often associated not to constitute a causative factor. Some people appear to be relatively immune to any or all of these causes, at least so far as continuing or annoying tinnitus is concerned. Many persons have no tinnitus even under extreme provocation, while others under little provocation suffer from marked tinnitus. In other words, there is pronounced idiosyncrasy or differing sensitivity in persons as to the degree of irritation and the time necessary to cause tinnitus. One of the more constant causes or accompaniments of tinnitus

is arteriolosclerosis; yet this disease may be prominent and yet cause no constant or annoying symptoms. Like multiple sclerosis, it is known to be spotty at all ages. Even adjacent vessels often show noticeable variations in degree of involvement, so that old people, almost all of whom have arteriolosclerosis, may, in spite of it, suffer no tinnitus.

The mechanism of nonvibratory tinnitus may be roughly compared with the mechanism of itching. When the neural mechanism of the ear or that of the skin is sufficiently irritated by any intrinsic disturbance, there results an illusion of sensation. The sense organs of the ear and of the skin set up their specific nerve impulses which are transmitted to the brain to be interpreted according to the special sensory nerves involved. If these impulses originate from an end organ in the skin, the interpretation is that of touch, pressure, tickling, itching or burning; if they are from the ear, a sensation of sound, a tinnitus, or a sensation of disturbed equilibrium is noted, depending on whether the auditory or the vestibular mechanism is involved. Localized edema or blebs occurring in the skin and edema, abnormal endolymph pressure or hydrops in the labyrinth are identifiable causes of such irritations. Associated with all of these changes of pressure there is a vascular change which undoubtedly usually results in local anoxia. Local conditions may modify the degree and therefore the relative bilaterality of the symptoms.

In the presence of binaural irritations producing bilateral tinnitus similar in timbre and intensity, slight monaural manipulations may appear to intensify or to refer the tinnitus wholly to the right or the left ear for long or short intervals. Frequently the judgment is poor as to lateralization. Also it is influenced by the memory of a prior sensation lateralized to the right or to the left ear.

If there is a detectable difference in the quality of the tinnitus, irrespective of intensity, in the two ears, the bilaterality of the tinnitus is easily detected. But when the quality is the same in the two ears, bilaterality is not so easily detected, because a louder tinnitus of one ear tends to mask a fainter tinnitus of the other, and the patient may think that he has tinnitus only in one ear, whereas he really has it in both. It is then subaudible in the apparently silent ear. This is well brought out when after a fenestration operation or a division of the eighth nerve the tinnitus appears in the ear not operated on. Strangely enough, this is a rare occurrence.

A dominance of the tinnitus of one ear occurs in both vibratory and nonvibratory tinnitus and is brought about in some such manner as in the case in which a faint sound applied to one ear is easily masked by a louder similar sound heard by the opposite ear. This binaural differential of masking effect is one of the pitfalls in contralateral masking.

As with many other bodily symptoms, one is frequently unable to determine definitely the cause of a tinnitus or even its exact place or

places of origin. When this is the case one must set up a hypothesis to enable one to deal intelligently with the problem.

In all sense organs there are subthreshold (not sensed) as well as overthreshold (sensed) irritations. There are subaudible and audible actual environmental stimulations, and subaudible and audible biologic stimulations.

When a nonvibratory tinnitus subsides, the lesions causing it have not necessarily wholly disappeared; they may still be present but of insufficient severity to make their presence known to the patient. They are then subaudible. There seems little or no doubt that this frequently happens. It may at first thought appear that these conceptions are of purely academic interest, but I will show that such may not be the case.

Even though there is no infallible way to demonstrate that there are in the cochlea subaudible stimulations caused by actual sound or by biologic irritations, several observations confirmatory of this belief and several modes of approach may be listed:

1. In the first place every person with normal hearing will be aware of some sounds even in the absolute quiet of a well soundproofed room. These intrinsic sounds are otherwise subaudible.

2. In the presence of a sound vibration which is just below threshold a small amount of additional intensity of the same vibration is required for this to reach the threshold of sensation. The intensity required to bring the sound to threshold varies directly with its distance below threshold. With discrete frequencies, as with a band of frequencies, this is common knowledge and easily demonstrated.

3. In the presence of a drug-caused irritation insufficient to cause an audible tinnitus, one may superimpose an additional irritation by means of additional measured amounts of the same drug. Then one may compare the amount or dose required to bring the tinnitus to threshold or over in a given patient with the amount required to cause tinnitus in the normal subject, or the amount necessary to reach threshold in the same patient during different phases of his tinnitus. In this way one may devise a means of measuring the dose required for a given person to increase the biologic stimulation sufficiently to bring the tinnitus into consciousness. Thus one has acquired a tool for determining the presence and the degree of the subaudible irritation and, therefore, a gage of the extent of the lesion etiologically associated with it. Such a test will, by trial and error, determine idiosyncrasy and the advisability of avoiding procedures which caused or increased the tinnitus. One also in this way may obtain information as to the place of origin of the tinnitus, because certain drugs differ in their affinities for different portions of the nerve and its end organ and in their patterns of stimulation or paralysis at different levels in the brain.

4. I have shown how audible tinnitus is affected by various masking sounds, and I see no reason why subaudible irritations may not cause effects which may be detected by close observation and experiment.[3] One such effect may be uncertainty in determining the threshold intensity of applied sounds. This uncertainty occurs too often (with and without audible tinnitus) in instances of nerve deaf-

3. Mr. R. L. Wegel and I have reported certain phenomena which seem to encourage further experimentation along these lines (Fowler [2a]).

ness, in which the threshold is otherwise definite indeed, to be a mere accident. This leads to the hypothesis that the presence of subaudible tinnitus, like the presence of audible tinnitus, tends to lessen the accuracy of the determination of threshold intensities. Comparing an ear in which there is audible tinnitus with an opposite normal ear which senses no tinnitus often demonstrates such a phenomenon clearly. The closer the test sound approaches the frequency of the tinnitus, and generally the louder the tinnitus, the more uncertain the threshold of the ear with tinnitus becomes. The degree of uncertainty may vary with the frequency location of the lesion and with the degree of the irritation.

5. Another approach is to estimate the amount and the time of pressure applied on the vessels of the neck or on the carotid plexus required to diminish or increase tinnitus or to bring subaudible tinnitus into consciousness. This procedure sometimes produces results which may be useful in treatment. The mere demonstration that the tinnitus can be changed in quality or intensity is welcome to the patient and encouraging to the otologist.

6. A routine experiment is to note the effect on tinnitus when positive and negative air pressures are produced in the external auditory canal. Either positive or negative pressure may cause tinnitus, or increase or diminish tinnitus already present. Negative results, in the absence of conduction deafness, suggest involvement of the central neurons. Tending to confirm this is the fact that changes of pressure occurring in the external auditory meatus appear to have no effect on tinnitus that is difficult to mask. This would be expected if this type of tinnitus originates central to the end organs.

7. Still another approach is to determine whether slight or large psychologic upsets are required to bring back, increase or change the tinnitus in any way. If they have no effect, the emotional factor probably plays a minor, if any, etiologic part. This information must be obtained from a careful history and from some well timed emotion-provoking questions. It is only a qualitative test but one often useful in diagnosis and treatment.

There are several other factual and theoretic approaches to the study of tinnitus, but I shall not go into them at this time.

Unfortunately, no one comes to the otologist for the prevention of tinnitus unless he is actually suffering from it or has recently suffered from it severely. However, if in any patient there has been a history even of long past tinnitus, one should take advantage of this evidence to search for the presence of lesions producing subaudible irritations, in order to study them and to institute preventive treatment.

A satisfactory approach to treatment may be briefly outlined as follows:

1. Hold out no "sure cure." There is none.

2. Obtain a complete past and present family and personal history. Include in this the endocrine, emotional and traumatic episodes, the infections, the circulatory disorders and the neurologic backgrounds. Record all of the drugs and other treatments which the patient has received and the results of treatment, if any. Stop the use of those which are not indicated. The greatest offenders are usually the barbiturates, acetylsalicylic acid and salicylates, and opiates generally. Many drugs commonly used to diminish tinnitus may actually increase it. When this is the case, a drug with the opposite action is indicated. I will mention one

example: When nicotinic acid, syntropan, atropine or any of the belladonna group make the tinnitus worse, switch to a drug of opposite action; give physostigmine or some other like acting drug. In large dosage some vitamins may do harm; in moderate dosage, none. In my experience they do little or no good except as placebos unless definitely indicated. What the patients need is not "dope," but relief from "dope" of all kinds whether it be from drugs, habits, thoughts or so-called "psychosomatic reactions." The history form should be so constructed that it includes all avenues of approach to diagnosis and treatment.

3. Make a thorough otologic examination, including audiometric tests at and above threshold.

Estimate or, better, carefully measure the frequency and the intensity of the tinnitus by having the patient compare and balance its loudness and tone with the loudness and tone of similar testing sounds. Take advantage of periods when the tinnitus is present to obtain through these means a tool for exploring periods when it is subaudible. (The otologist and the patient will often be surprised to find that the real intensity of the tinnitus is slight.) Apply the tests discussed in this paper and explore for subaudible tinnitus along the lines I have suggested. If the tinnitus is bilateral and especially if equally bilateral, it is most important to study the whole body as well as the ear. The determining factor of tinnitus may lie almost anywhere.

5. Determine the time of day and the coincidental environmental factors and cycles associated with variations in the loudness and the timbre of the tinnitus. Do not neglect vasomotor and allergic factors. Here diphenhydramine hydrochloride ("benadryl hydrochloride") is of service in many instances. If the tinnitus varies from hour to hour or at longer intervals, tell the patient that this is a favorable sign—because it frequently is a favorable sign. It indicates that the lesions are not wholly irreversible. The otologist should search for the cause of their variability. Even constant tinnitus varies in intensity at times, and this ebb and flow should be measured and utilized in diagnosis and treatment.

6. Make laboratory tests when indicated, particularly concerning the hemoglobin, the serologic status, the basal metabolism, the blood chemistry (sometimes the level of folic acid), make total and differential blood counts, determine the erythrocyte sedimentation rate and make such other determinations as are indicated.

7. Search for dysfunction or distress in all regions of the body. Blood pressure is important, particularly changes in blood pressure. Check it frequently. Do not neglect the teeth. Abscess and changes of the bite are known causes of tinnitus.

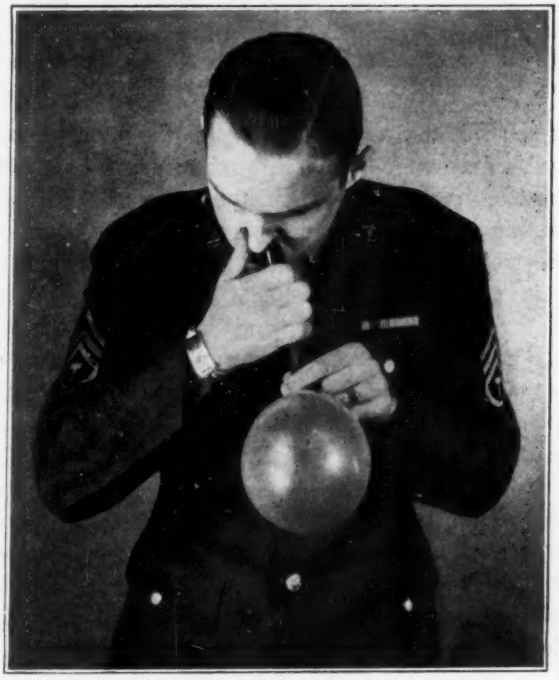
8. Determine dietary and living habits—eating, drug-taking, drinking and smoking habits, including even the consumption of such common things as salt, coffee and water, which may be excessive. Dig deep to uncover the truth.

9. Office politzerization, even when indicated, is of limited use because it is usually administered at too long intervals. Routine catheterization of the eustachian tubes often does more harm than good and is seldom indicated in the treatment of the lesions causing nonvibratory tinnitus. The improvement of hearing often reported is not substantiated by proper audiograms and generally reflects merely the projection of a wish of both patient and otologist.

If the patient really needs inflation of the middle ear, and politzerization can be successfully carried out (in most cases it can be), I have the patient supplement office treatment with gentle inflations at home, using a soft balloon bag, the Fowler ear inflator (figure), which has served me well for the past forty years.

See to it that for a few weeks the inflater is used at least four times a day and that half a dozen inflations are given at each sitting. For easy remembrance I usually prescribe politzerization as follows: "on arising in the morning, before retiring at night, and before and after each meal." This will total eight times a day at home, whereas the general practice is to inflate only once a week or at most two or three times a week. How can one expect to reduce edema or chronic mucoid blockage of the eustachian tube adequately and permanently by such inadequate treatment?

With children generally, and with some adults, when there is hypertrophied lymphoid tissue in the nasopharynx, an application of radium or of radon is beneficial. Beware of over-roentgenization. It makes for dry throats, and there is always the danger of producing a neoplasm.



The customary method of using the Fowler inflater: The patient breathes into the balloon gently through the nose so as to supply it with sufficient air to be compressed synchronously with the act of swallowing.

10. Uncover any emotional factors underlying or influencing the tinnitus directly or indirectly, with sympathetic understanding. This is an important measure in diagnosis and treatment. Marital, family and sexual maladjustments, frustrations and obsessions, as well as menstruation and the menopause (natural or artificial), are often potent factors in bringing tinnitus above threshold.

11. Explain to the patient in simple words how tinnitus may be affected by all of these factors. Instruct him how to lessen or remove them. This will take away some of the mystery, or appear to do so, and allay apprehensions of impending calamity.

12. Never tell a patient that nothing can be done for him. This is the expression of a defeatist attitude and is bad psychotherapy; moreover, it is not true if the otologist is sincerely interested in helping him. In the majority of instances tinnitus can be brought below, or to so little over, the threshold of hearing that it is no longer a major irritation. There are few instances in which one cannot at least lessen the tinnitus to some extent, and fewer still in which, even though the tinnitus persists, one cannot help the patient to lose his exaggeration of it and his fear of "going deaf" or "going insane." Tell him that tinnitus never made any one insane but that insane people may have tinnitus. Assure the patient that the tinnitus alone will not make him deaf, because per se it will not make him deaf. It is the lesion causing the irritation (audible and subaudible) and not the tinnitus itself which threatens further loss of hearing. Explain that such lesions may often be controlled to a point where they cause little or no damage and where the irritations causing the tinnitus are made subaudible. Jolt the patient out of his habit of bewailing his fate and feeling much too sorry for himself. Send him out of the office laughing at himself and his troubles. Make him realize that his tinnitus is not actually as loud in his ear as in his mind.

I am sure that otologists would welcome the name of some one drug or surgical procedure which will "cure" tinnitus. There is not now and never will be one drug or surgical method that will do this. (There is no single cause.) Drugs alone or operations alone usually fail. Even operations destroying the nerve or the labyrinth in which a severe loss of hearing is already present should be advocated with restraint. Removal of tonsils and adenoids and resection of the nasal septum rarely if ever alleviate tinnitus. It is inadvisable even to suggest to a patient that there is an operation to stop tinnitus. Once this has been suggested, the psychotic patient will insist on having it done,, and when in such cases it fails, especially if the deafness and the tinnitus increase, a fixation or obsession is acquired which will surely need the attention of a psychiatrist. Every patient as well as his tinnitus must be treated individually; no two will respond alike. Nonvibratory tinnitus is a problem that involves more than the ear. Time and effort spent in exploring along the paths I have suggested will, I believe, be well worth while. Two closing suggestions: Do not neglect the lesions causing the deafness. Nothing is more effective in curing tinnitus than diminishing deafness and nothing so harmful as increasing deafness. Do not transfer the responsibility of the otologist to some neurologist, psychologist or brain surgeon. Few such are sufficiently interested in the otologic and personal problems involved to be of service except as consultants in their special fields. In this field of endeavor continuing sympathetic understanding and the cooperation of all concerned are imperative if one expects to obtain results. I trust my facts and fancies will serve to stimulate some who read this to a greater interest in the problems of tinnitus.

140 East Fifty-Fourth Street.

NASAL HEMORRHAGE

Studies of Ascorbic Acid, Prothrombin and Vitamin K

HARRY NEIVERT, M.D.
AND
RECHA ENGELBERG, Ph.D.
NEW YORK
AND
LEO A. PIRK, Ph.D.
NUTLEY, N. J.

NOSEBLEED may be due to a large number of widely diversified conditions. Epistaxis is labeled "idiopathic hemorrhage" once the local and systemic causes generally recognized as precipitating or conducive factors are excluded. However, such a "diagnosis" is unsatisfactory and does not lead to causal therapeutic measures. Therefore, it was decided to investigate whether hitherto unrecognized etiologic factors could not be detected.

In the course of investigations on the causes of late tonsillar hemorrhage it was demonstrated that prothrombin or ascorbic acid (vitamin C) deficiencies are important contributing factors.[1] These findings prompted us to study ascorbic acid and prothrombin levels in cases of epistaxis. Some workers have casually suggested the administration of ascorbic acid in nosebleed. For instance, Jones [2] stated that "C [ascorbic acid] may be indicated in some cases of epistaxis," and Nelson [3] expressed his "belief" that many cases of nasal hemorrhage "have as their background a subclinical scurvy." However, neither of them determined ascorbic acid levels, nor did they or any other investigator estimate the prothrombin activity of patients suffering from epistaxis.

From the Pharyngology Research Laboratory, Department of Otolaryngology, Columbia University College of Physicians and Surgeons, and Presbyterian Hospital, New York, and Hoffmann-La Roche, Inc., Nutley, N. J.

1. Neivert, H.: Late Secondary Tonsillar Hemorrhage: I. Studies of Prothrombin and Vitamin K, Arch. Otolaryng. **42**:14-18 (July) 1945. Neivert, H.; Pirk, L. A., and Engelberg, R.: Late Secondary Tonsillar Hemorrhage: II. Studies of Ascorbic Acid, ibid. **43**:568-577 (June) 1946.

2. Jones, I. H.: Vitamin Therapy Today, Laryngoscope **52**:805-814 (Oct.) 1942.

3. Nelson, L. A., in discussion on Windham, R. E.: Cause and Treatment of Rhinopharyngeal Hemorrhages, Texas State J. Med. **40**:484-487 (Jan.) 1945.

CASE MATERIAL, PROCEDURE AND FINDINGS

The present series consists of 104 consecutive patients presenting themselves with nosebleed at Presbyterian Hospital, New York. As may be seen from the table, there were 59 males, ranging in age from 10 to 77 years, and 45 females, whose ages ranged from 11 to 72 years. Five patients of the entire group had recently undergone nasal operations, and in them the epistaxis may be classified as late postoperative bleeding. In 7 other patients the hemorrhages may have had origin also in some local cause. Thus 1 patient had a diagnosis of intramucosal squamous cell epithelioma; 3 patients were suffering from chronic maxillary sinusitis; 2 patients had nasal allergy, and 1 an infection of the upper respiratory tract.

In 45 instances the nasal hemorrhages were associated with disease of the blood vessels, with blood dyscrasias, with infectious disease or with other constitutional diseases which may give rise to abnormal bleeding. Thus, 29 patients had hypertension and 1 arteriosclerosis. There were 7 who showed anemia, a condition which may have precipitated the hemorrhage (8 other patients also had anemia coincidental with some other etiologic factor). One case was diagnosed as thrombopenic purpura, and 2 as hereditary telangiectasia. One patient suffered from pulmonary tuberculosis; another, from rheumatic fever. Gaucher's disease (xanthomatosis), amyloidosis of the liver and kidneys and Hodgkin's disease (lymphogranulomatosis) were diagnosed in 3 cases, respectively.

This leaves 47 cases without any apparent etiologic factor for the hemorrhage.

Generally, the nosebleed was severe, necessitating blood transfusions in 19 cases. In one of these the blood loss was so great that six transfusions had to be given during one period of hospitalization. In several other cases repeated blood transfusions were required.

Forty-eight patients complained of recurring episodes of bleeding, and some had experienced intermittent nasal hemorrhages for many years. For instance, a patient with hereditary telangiectasia (case 67) had frequent nosebleed for twenty years, with one to two hemorrhages occurring daily during the three years prior to his examination. Such a history may be expected from a patient with a severe hemorrhagic disease. In contrast, there was no blood dyscrasia in patient G. H. (case 94), and yet she had suffered from frequent nasal hemorrhages at irregular intervals for eighteen years prior to her examination.

Ascorbic acid and prothrombin plasma levels were determined for all 104 patients. Those in whom the underlying pathologic process seemed evident were not excluded, because it was suspected that ascorbic acid and/or prothrombin deficiency superimposed on the predominant etiologic factor might increase the number of episodes of bleeding or add to their severity.

In carrying out the estimations of ascorbic acid, the method of Mindlin and Butler[4] was used. According to this technic, the decrease in the concentration of oxidized indophenol produced by the addition of an amount of plasma which is insufficient to cause complete reduction of the dye is measured by a photoelectric colorimeter.

---

4. Mindlin, R. L., and Butler, A. M.: The Determination of Ascorbic Acid in Plasma: Macromethod and Micromethod, J. Biol. Chem. **122**:673-686 (Feb.) 1938.

Blood plasma ascorbic acid values of 0.5 mg. per 100 cc. or below were chosen arbitrarily as reflecting a state of ascorbic acid deficiency.

In the assays of prothrombin, the method first described by Fullerton [5] and advocated by Page and his associates [6] was used. According to this technic, viper venom [7] and calcium chloride are added to plasma obtained by centrifuging the decalcified venous blood. The sharp end point is characterized by formation of fibrin particles, which are clearly visible in the agitated test tube.

A prothrombin time of 25 seconds or more was taken to indicate a prothrombinopenic state.

As appears from the table, which lists the averages of the duplicate determinations performed, there were only 11 instances, or 10.57 per cent, in which both the plasma ascorbic acid and the prothrombin level were normal. The concentration of plasma ascorbic acid was below normal in 68 patients, an incidence of abnormality of 65.38 per cent. (In 22 subjects the plasma ascorbic acid ranged from 0 to 0.10 mg., in 28 from 0.11 to 0.30 mg. and in 18 from 0.31 to 0.50 mg., per 100 cc.) Of these 68 patients with the aforementioned hypovitaminosis, 35, or 33.65 per cent of the total group of 104, also showed prothrombin deficiency. There were 25 patients, or 24.03 per cent, who were normal with respect to their plasma ascorbic acid but whose prothrombin activity was subnormal. These findings are shown graphically in the chart.

In the group of 47 patients with no apparent underlying cause for the hemorrhage there were 4 whose ascorbic acid and prothrombin levels were normal—an incidence of normality of 8.51 per cent. On the other hand, in the group of 57 patients whose nosebleeds were traceable to one of the generally recognized local or systemic causes, there were 7, or 12.28 per cent, in whom both ascorbic acid and prothrombin levels were normal. Thus, from the point of view of percentage there were fewer patients with both a normal ascorbic acid and a normal prothrombin level in the group in which there was no apparent background for the bleeding. Yet, the incidence of this hypovitaminosis and of hypoprothrombinemia was striking in both groups.

---

5. Fullerton, H. W.: Estimation of Prothrombin: A Simplified Method, Lancet **2**:195-196 (Aug. 17) 1940.

6 Page, R. C., and Russell, H. K.: Prothrombin Estimation Using Russell Viper Venom: I. Simple Modification of Quick's Method, J. Lab. & Clin. Med. **26**:1366-1370 (May) 1941. Page, R. C.; DeBeer, E. J., and Orr, M. L.: Prothrombin Studies Using Russell Viper Venom: II. Relation of Clotting Time to Prothrombin Concentration in Human Plasma, ibid. **27**:197-201 (Nov.) 1941; III. Effect of Lecithinized Venom on Prothrombin Clotting Time, ibid. **27**:830-834 (March) 1942. Page, R. C.; DeBeer, E. J., and Bercovitz, Z.: Prothrombin Studies Using Russell Viper Venom: IV. Relation Between Prothrombin Clotting Time and Plasma Fibrinogen Concentration, ibid. **28**:910-912 (April) 1943. Page, R. C., and DeBeer, E. J.: Prothrombin Studies Using Russell Viper Venom: V. Interbatch Variations of Venom, ibid. **28**:912-915 (April) 1943; VI. The Stability of the Thromboplastic-Like Activity of Russell Viper Venom Under Various Conditions of Storage, Am. J. M. Sc. **206**:336-339 (Sept.) 1943.

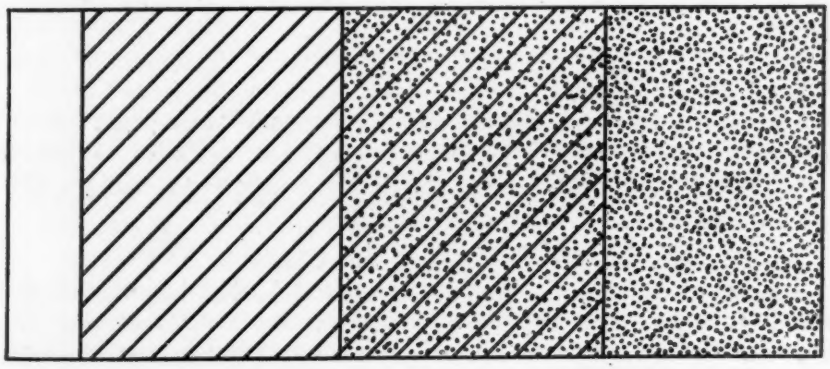
7. Dr. D. S. Searle, of Burroughs-Wellcome, Inc., New York, furnished generous supplies of Russell viper venom "Stypven."

Blood Plasma Level of Ascorbic Acid and Prothrombin Time as Determined in 104 Consecutive Cases of Epistaxis

Case	Patient	Sex	Age	Ascorbic Acid Level: Above 0.5 Mg. per 100 Cc.	Ascorbic Acid Level: 0.5 Mg. or Less per 100 Cc.	Prothrombin Time: 25 Seconds or Above	Prothrombin Time: Below 25 Seconds	Comment
1	K. I.	F	56	....	0.42	....	24.0	Anemia
2	G. I.	F	30	....	0.25	....	22.3	Chronic maxillary sinusitis
3	E. I.	F	43	....	0.43	29.9	....	Arteriosclerosis
4	A. A.	F	31	....	0	30.9	....	
5	N. K.	M	30	....	0.15	26.8	....	Anemia
6	D. C.	M	45	....	0.22	29.2	....	Anemia
7	F. A.	F	47	....	0.08	35.2	....	Pulmonary tuberculosis
8	C. S.	F	21	....	0	30.1	....	Thrombopenic purpura
9	H. F.	F	66	....	0.24	....	19.0	Hypertension
10	L. T.	M	71	....	0.12	26.2	....	Hypertension
11	W. W.	M	77	0.54	....	25.0	....	
12	T. B.	M	33	....	0.14	....	18.8	Rheumatic fever
13	Q. B.	M	56	....	0	....	24.5	Hypertension
14	F. H.	M	15	1.0	....	27.8	....	
15	F. S.	M	53	....	0.32	....	19.0	Chronic maxillary sinusitis
16	L. J.	M	45	....	0.15	....	22.6	Hypertension
17	F. M.	M	16	....	0.24	....	22.0	
18	S. C.	M	57	....	0.3	....	18.0	
19	S. M.	F	60	....	0.15	25.0	....	Hypertension
20	B. M.	M	43	....	0	25.0	....	Hypertension
21	P. A.	M	64	....	0	....	18.6	
22	S. M.	F	54	....	0	25.6	....	Hypertension
23	M. T.	M	29	1.3	....	....	23.4	Nasal allergy
24	M. E.	F	72	....	0.4	....	21.4	
25	K. M.	M	43	....	0.39	....	20.0	
26	F. G.	M	42	....	0.20	....	24.0	
27	K. H.	M	45	....	0.18	29.2	....	Anemia
28	J. M.	F	45	0.8	....	30.0	....	
29	L. E.	F	60	....	0.3	29.8	....	Hypertension
30	B. M.	F	53	....	0.42	26.0	....	Hypertension
31	L. J.	M	43	....	0.25	28.6	....	Anemia
32	P. J.	M	51	0.55	....	....	19.6	Hypertension
33	K. C.	M	54	....	0.05	....	22.5	Hereditary telangiectasia
34	M. J.	M	35	....	0.1	....	18.0	
35	F. C.	F	56	....	0.3	26.0	....	
36	C. E.	M	45	....	0.06	25.0	....	Infection of the upper respiratory tract
37	H. E.	F	35	....	0.3	....	24.8	
38	S. B.	M	33	....	0.3	....	19.2	
39	M. D.	F	49	0.7	....	....	22.0	Hypertension
40	H. F.	F	28	....	0.27	....	24.6	Anemia
41	P. F.	M	56	....	0	....	23.0	
42	Q. M.	F	35	....	0.3	25.0	....	Nasal allergy
43	F. A.	M	68	....	0.1	....	23.2	
44	O. A.	M	49	....	0.05	26.5	....	
45	G. M.	F	40	1.2	....	....	22.5	
46	S. M.	M	18	1.2	....	25.0	....	Submucous resection 2 weeks before epistaxis
47	K. M.	M	27	....	0.06	60.2	....	Gaucher's disease of spleen and liver
48	D. S.	F	58	....	0.3	39.0	....	Amyloidosis of liver and kidneys
49	O. T.	M	11	....	0	....	23.0	
50	K. M.	M	42	....	0.1	....	21.6	Submucous resection 1 week before epistaxis
51	M. C.	F	45	....	0.5	....	19.6	
52	F. P.	M	38	....	0.2	....	22.0	
53	S. W.	M	10	0.58	....	....	23.0	
54	F. S.	M	45	1.1	....	....	23.4	
55	I. S.	M	34	1.3	....	26.0	....	Submucous resection 10 days before epistaxis
56	S. A.	M	60	....	0.17	....	22.2	
57	K. L.	M	67	....	0.4	29.5	....	Hypertension
58	B. C.	F	17	0.6	....	32.0	....	30 grains (2 Gm.) acetylsalicylic acid daily for several days
59	K. E.	F	45	1.0	....	27.5	....	Hypertension
60	H. M.	F	42	....	0.4	....	23.2	Hypertension
61	M. C.	F	15	1.2	....	25.0	....	
62	F. S.	F	41	0.7	....	25.0	....	Hypertension
63	G. J.	M	48	....	0.39	....	18.8	
64	H. R.	M	25	0.9	....	....	21.0	Submucous resection 1 week before epistaxis
65	T. J.	M	46	....	0.28	....	19.6	
66	H. J.	M	57	....	0.50	....	21.2	
67	H. T.	M	40	....	0.17	25.6	....	Hereditary telangiectasia

Blood Plasma Level of Ascorbic Acid and Prothrombin Time as Determined in 104 Consecutive Cases of Epistaxis—Continued

Case	Patient	Sex	Age	Ascorbic Acid Level: Above 0.5 Mg. per 100 Cc.	Ascorbic Acid Level: 0.5 Mg. or Less per 100 Cc.	Prothrombin Time: 25 Seconds or Above	Prothrombin Time: Below 25 Seconds	Comment
68	J. N.	F	49	0.75	....	27.0	....	Chronic maxillary sinusitis
69	W. A.	M	60	0.86	....	29.0	....	
70	St. F.	F	71	1.0	....	....	20.0	Hypertension
71	N. S.	M	68	....	0.06	29.0	....	Hypertension
72	St. H.	F	49	0.8	....	27.0	....	Hypertension
73	A. F.	F	66	....	0	30.4	....	Hypertension
74	K. R.	F	43	....	0.49	31.5	....	Hypertension
75	B. K.	F	53	1.0	....	33.0	....	Hypertension
76	W. A.	F	42	0.9	....	27.9	....	Hypertension
77	G. E.	F	11	1.5	....	29.9	....	
78	M. O.	M	38	....	0.18	32.5	....	
79	W. V.	F	19	....	0.33	28.0	....	
80	H. S.	M	17	1.1	....	25.5	....	
81	G. L.	M	28	1.0	....	31.0	....	
82	A. M.	M	43	1.0	....	....	24.0	
83	C. R.	M	54	....	0.18	33.4	....	Hypertension
84	S. M.	M	67	0.59	....	28.4	....	Hypertension
85	H. G.	M	23	....	0.2	31.0	....	Submucous resection 1 week before epistaxis
86	M. D.	F	45	1.1	....	30.3	....	Anemia
87	J. B.	F	46	0.78	....	25.3	....	
88	B. A.	M	24	....	0	....	24.1	
89	G. M.	M	51	0.78	....	....	24.0	Hypertension
90	L. J.	M	53	0.98	....	32.3	....	
91	G. O.	F	59	....	0.05	28.6	....	Hypertension
92	D. L.	F	12	0.7	....	31.5	....	
93	J. H.	M	14	1.0	....	30.6	....	
94	G. H.	F	31	....	0.5	26.0	....	
95	S. O.	F	29	....	0.06	40.0	....	
96	S. J.	M	28	....	0.42	....	24.0	
97	S. H.	M	24	....	0.50	26.0	....	
98	G. R.	F	69	....	0.39	25.0	....	Intramucosal squamous cell epithelioma
99	C. A.	F	65	1.0	....	....	23.8	Hypertension
100	K. C.	F	71	....	0.39	....	23.9	Hypertension
101	A. R.	M	27	....	0	62.0	....	Hodgkin's disease
102	H. G.	F	37	0.85	....	25.0	....	Hypertension
103	P. H.	M	33	0.96	....	25.0	....	
104	K. G.	M	60	....	0.13	....	19.0	



Incidence of blood plasma ascorbic acid deficiency and prothrombinopenia in 104 consecutive cases of epistaxis.

The white column represents 11 patients with normal levels of blood plasma ascorbic acid and normal prothrombin times.

The cross-hatched column represents 33 patients with subnormal ascorbic acid levels and normal prothrombin times.

The cross-hatched and dotted column represents 35 patients with subnormal ascorbic acid levels and prothrombinopenia.

The dotted column represents 25 patients with normal ascorbic acid levels and prothrombinopenia.

THERAPEUTIC MEASURES

Therefore, once these deficiencies became apparent, it was decided to institute corrective measures which would have been indicated even without hemorrhages. As may be seen from the foregoing observations, 93 patients of the total series of 104 were deficient in ascorbic acid and/or prothrombin, but for reasons beyond our control substitution therapy could be initiated in 62 patients only. Of these, 15 were given ascorbic acid,[8] 9 a vitamin-K-like compound ("synkayvite") [8] and 38 persons both vitamins.[8] However, not all of the latter patients had been shown to have a deficiency of both the vitamins they received. With some deviations, the daily dose was 300 mg. of ascorbic acid or 15 mg. of "synkayvite" or a combination of these amounts. In general, medication was given for one to two weeks. This was mostly sufficient to overcome a hypovitaminosis, and in some instances this course was repeated later if reexamination revealed a recurrence of the deficiency.

The follow-up of the 62 patients receiving vitamin therapy was extremely unsatisfactory, with 26 failing to report back to the clinic as requested. Among the latter were several patients who had repeatedly sought medical advice at the ear, nose and throat department of the Presbyterian Hospital for many years, whenever troubled by a hemorrhage. This suggests that they derived some benefit from the vitamin therapy. Of the 36 patients whose response to treatment could be determined, 27 showed clinical improvement ranging from complete cessation to decreased frequency of epistaxis and 9 showed no improvement. It is noteworthy that among those who undoubtedly derived some benefit from the vitamin therapy were several hypertensive patients with ascorbic acid deficiency and/or prothrombinopenia as a concomitant finding.

Since most of those who did return for reexamination could not be induced to come back at regular intervals for ascorbic acid and prothrombin determinations, the correlation of laboratory findings and clinical course was not achieved in all cases.

A few case histories illustrative of this correlation follow:

CASE 28.—J. M., A woman aged 45, complained of repeated daily nasal hemorrhages. Nasopharyngeal and general medical examination did not reveal any abnormalities. Temporary stoppage of bleeding was effected by nasal packings. A determination of blood plasma ascorbic acid yielded 0.8 mg. per 100 cc.—a very satisfactory value. However, estimation of the prothrombin reflected a lowered

8. Hoffmann-La Roche, Inc., Nutley, N. J., furnished generous supplies of "vitamin C 'Roche' tablets (100 mg.)," "'synkayvite' tablets (5 mg.)" and combined experimental tablets (not generally available) which contain 100 mg. of ascorbic acid and 5 mg. of "synkayvite," a water-soluble vitamin-K-like substance (tetrasodium 2-methyl-1,4-naphthohydroquinone diphosphoric acid ester).

level (the prothrombin time was 30 seconds). For two days more the patient was kept under observation without corrective measures, and the daily episodes of bleeding continued. Then 15 mg. of the vitamin-K-like preparation, "synkayvite," was administered daily for two weeks, and this resulted in a decrease of the prothrombin time to 21 seconds after five days. One week after discontinuation of "synkayvite" therapy there was another episode of bleeding. Reexamination of the prothrombin time showed an increase to 30 seconds, reflecting a state of prothrombin deficiency. Administration of "synkayvite" was reinstituted at a dosage level of 15 mg. a day. The prothrombin time came down promptly to 21 seconds and remained well below the upper normal level of 25 seconds during the subsequent twelve weeks during which the patient continued to take the vitamin-K-like drug. There were no episodes of bleeding during this time. She was then advised to substitute liberal amounts of green salad and vegetables for the vitamin-K-like drug, and during the eighteen months that intervened between this change of therapy and the time when she was last heard of, there was no nosebleed. Eight months after the institution of this dietary regimen an assay of prothrombin showed prothrombin activity somewhat lower than during the period of supplementation with "synkayvite," but the prothrombin time (24 seconds) was still below the upper limit of normal.

CASE 94.—G. H., a woman aged 31, had been suffering from frequent nasal hemorrhages for eighteen years. The nosebleed occurred at irregular intervals, but during the past four years there was an episode of bleeding at least every other week. All clinical findings were normal. Determination of the blood plasma ascorbic acid and estimation of the prothrombin content revealed mild deficiency of both ascorbic acid and prothrombin (the findings were 0.5 mg. of ascorbic acid per 100 cc. and a prothrombin time of 26 seconds). A daily dose of 15 mg. of "synkayvite" and 300 mg. of ascorbic acid, given combined in three tablets, was administered for one week. At that time reexamination of the ascorbic acid level showed an increase to 0.9 mg. per 100 cc. of blood plasma, and the prothrombin time was 25 seconds. There were no episodes of bleeding. Five weeks after discontinuation of this medication the prothrombin time had risen to 27 seconds and the ascorbic acid concentration was 0.7 mg. per 100 cc. While such changes may not be significant, the administration of the tablets containing both ascorbic acid and "synkayvite" was reinstituted for one week. This resulted in a rise of ascorbic acid to 1.05 mg. per 100 cc. and a reduction of prothrombin time to 24.5 seconds. Two and one-half months later the prothrombin time was 21.8 seconds and the ascorbic acid plasma level 0.6 mg. per 100 cc. When this patient was last heard of she had not had an episode of bleeding for over six months—a truly remarkable improvement considering her past history.

CASE 75.—B. K., a woman aged 53, was troubled by frequent nasal hemorrhages for seven years. When first seen her blood pressure was 180 systolic and 80 diastolic, and a diagnosis of essential hypertension was made.

Determination of blood plasma ascorbic acid revealed a level of 1 mg. per 100 cc. Estimation of prothrombin indicated a deficiency of this clotting factor (the prothrombin time was 33 seconds). The hypertensive heart disease was probably the main cause of the nasal hemorrhages, yet the prothrombin deficiency was looked on as a possible aggravating factor. Therefore, therapy was instituted in the form of "synkayvite"-ascorbic acid tablets. The patient thus received a daily dose of 15 mg. of the vitamin-K-like substance and 300 mg. of ascorbic acid for one week. At that time reexamination of prothrombin time showed no improvement. Therefore, medication was continued for another week; at the end of

this time the plasma ascorbic acid level was 1.4 mg. per 100 cc., but the prothrombin time had risen further, to 40 seconds, reflecting a marked deficiency of prothrombin. After two and a half months the patient was readmitted to the hospital because of another episode of severe bleeding. Although she received twice the former daily dose of "synkayvite," the prothrombin time after four days of medication was still 33 seconds. Thirty milligrams of "synkayvite" given daily for eight more days proved ineffectual in decreasing the prothrombin time, which was then 35 seconds. There were almost daily nasal hemorrhages, necessitating a ligation of the external carotid artery. Two months later the patient was again admitted because of epistaxis. "Synkayvite" medication was reinstituted, and the inferior turbinate was removed. Blood transfusions had to be administered, and these brought her prothrombin time down to a normal value of 21.9 seconds. She was discharged in fairly good condition. In this case of epistaxis associated with hypertension and hypoprothrombinemia, the administration of the vitamin-K-like substance was not attended by improvement of nosebleed.

CASE 84.—S. M., a man aged 67, had suffered from frequent nasal hemorrhages for ten years. He had had an essential type of hypertension for several years. Estimation of prothrombin (28.4 seconds) revealed a deficiency of prothrombin and the blood plasma ascorbic acid level (0.59 mg. per 100 cc.) was just above the lower normal limit. A daily dose of 15 mg. of "synkayvite" and 300 mg. of ascorbic acid was prescribed for one week. At the end of that time the plasma ascorbic acid concentration showed an increase to 1 mg. per 100 cc. and the prothrombin time had come down to 24 seconds. Determinations of ascorbic acid level and prothrombin time, four weeks later, yielded normal values. After one more month without medication examination again revealed hypoprothrombinemia and normal concentration of ascorbic acid (the findings were a prothrombin time of 30 seconds and a plasma ascorbic acid level of 0.9 mg. per 100 cc.). Combined "synkayvite" and ascorbic acid therapy was reinstituted for one week. This resulted in a reduction of prothrombin time to 26 seconds. The patient was advised to substitute liberal amounts of green salad and vegetables for the vitamin-K-like drug. After two months on this dietary regimen his prothrombin time was 25.4 seconds and the plasma ascorbic acid concentration was 0.9 mg. per 100 cc. On reexamination two and a half months later there was no appreciable change in these values. In this patient, also, the hypertension was probably the underlying factor which precipitated the nasal hemorrhages. Yet the partial correction of a concomitant prothrombin deficiency and the increase of the plasma ascorbic acid resulted in complete control of his frequent episodes of bleeding for ten months—the period of observation.

SUMMARY AND CONCLUSIONS

One hundred and four patients suffering from epistaxis were studied and the plasma ascorbic acid levels and prothrombin times determined in all. The concentration of ascorbic acid and the prothrombin time were normal in only 11 of these patients, or 10.57 per cent. The plasma ascorbic acid level was subnormal in 68 patients, an incidence of 65.38 per cent. Of these patients, 35 also showed prothrombinopenia. There were 25 patients, or 24.03 per cent, whose plasma ascorbic acid concentration was normal but whose prothrombin activity was subnormal. Thus, a total of 60 persons, or 57.7 per cent, exhibited a hypoprothrombinemic state.

In 57 patients the nosebleed was traceable to one of the generally recognized local or systemic causes, and in 47 patients it was not. The percentage with both ascorbic acid and prothrombin findings normal was somewhat greater in the former group.

Vitamin therapy was instituted in 62 patients of the 93 found to be deficient in ascorbic acid and/or prothrombin. The clinical response could be determined in only 36 patients. Of these, 27 were definitely improved. Owing to factors beyond our control, laboratory findings could not be correlated with cessation of epistaxis or with less frequent occurrence of episodes of bleeding in all cases. Illustrative cases in which this correlation was accomplished are presented.

The extremely high incidence of ascorbic acid and prothrombin deficiency in a series of 104 consecutive cases of epistaxis and the gratifying clinical results attending the administration of ascorbic acid and/or a vitamin-K-like substance in some of these cases suggest that vitamins C and K have a definite place in the management of nasal hemorrhage.

CONGENITAL LARYNGEAL WEB: ITS ERADICATION

DAVID L. POE, M.D.
AND
PAUL S. SEAGER, M.D.
NEW YORK

THE SURGICAL eradication of laryngeal web tissue presently to be described represents a new procedure. Careful consideration of the literature does not reveal any similar work save, perhaps, Haslinger's.

Over twenty years ago one of us (Dr. Poe) was present when Haslinger performed an operation for the elimination of a soft tissue diaphragm, at which time Haslinger introduced a small metal plate between the vocal cords after the division of the web, and was afforded the opportunity to observe the patient during the course of convalescence until the latter was discharged as cured. Among the complications was the granulation tissue that formed on the raw surfaces of the cords, which required frequent applications of silver nitrate. Spells of coughing and general discomfort persisted until the delicate tissue of the upper respiratory tract became accustomed to the presence of the foreign body. The metal plate remained in place for about three weeks. Accomplished dexterity of a high order was required to carry out the demands of Haslinger's complete procedure. Reflecting on the large number of devices advocated for the destruction of a laryngeal obstacle of this type, we consider that Haslinger's comes closest to the one we are herewith recording. However, for the sake of clarity and to avoid confusion we believe it feasible for the present to omit controversial discussion as to the fundamental differences which exist between the two methods, nor do we wish to take up the discussion of other universally adopted procedures. We are of the opinion that the interest of the reader is best served if we confine ourselves to the one theme, namely, the presentation of our operation.

PRESENTATION OF A CASE

The patient was a white man, 19 years old, physically well developed. He could not recall any intercurrent ailments that in any way contributed to the laryngeal disability. His speech was high pitched when forced. He could not utter

From the Department of Otolaryngology, New York Post-Graduate Medical School and Hospital of Columbia University.

a loud sound. Attempts to emit one had no volume, and the voice did not carry beyond a normal loud whisper. His normal conversational tone was hoarse, non-vibrant and only slightly above the average whisper. He had had this kind of voice all his life. He avoided games requiring much physical exertion because they brought on breathing difficulties.

Laryngeal examination disclosed a shining white thin bit of tissue extending from one true vocal cord to the other, to both of which it was firmly and uninterruptedly attached. It extended from the anterior commissure slightly more than half the distance posteriorly. The free posterior edge presented a slightly concave outline.

When the patient came under our care, it was thought that perhaps dividing the diaphragm along the center with a sharp knife would be sufficient to free the cords and open a greater breathing area. It was considered worth trying since it was easy and entailed no risk. The operation was therefore carried out by one of us (Dr. Seager), and the patient was discharged from the hospital the following day. It was interesting to observe that as soon as the thin tissue was divided by the edge of a sharp knife, the respective halves instantaneously disappeared from view. There was no lax bit to be seen anywhere. For the moment it was thought that that was the end of the diaphragm. Three days later, when the patient presented himself for examination, the binding web was again seen to spread from cord to cord, appearing as though nothing had ever been done.

The new method proposed by one of us (Dr. Poe, who had successfully operated in 2 previous cases) was now undertaken.

Operation.—The interior of the larynx was anesthetized with cocaine. The skin and soft tissue over the cricothyroid junction and the anterior surface of the thyroid cartilage was anesthetized with an injection of a small amount of a 2 per cent solution of procaine hydrochloride. The larynx was exposed with a Haslinger directoscope. A tubular trocar designed especially for this work, with a sharp-pointed stylet, was pushed through from the outside into the interior of the larynx. The lower border of the thyroid cartilage was closely hugged by the instrument. The thin tissue forming the web was swabbed on its upper and lower surfaces with a 50 per cent solution of silver nitrate and then was divided in the center to the anterior commissure with a sharp knife. The trocar could be seen projecting in the midline just distal to the anterior commissure. The stylet was removed. A silver wire was then introduced into the larynx through the tube. As it appeared in the larynx it was grasped with forceps and brought out through the mouth. To its end a smooth metal (silver) plate was attached. A slow drawing of the other end of the wire, protruding from the trocar on the outside of the larynx, pulled the plate into the introitus. The plate, grasped in a long forceps, was guided through the laryngoscope into the area between the two cords as far as the anterior commissure. It was anchored in this position by fastening the wire on the outside of the larynx. Threading the wire through a button and over a gauze roll facilitated secure fastening.

The plate was held moderately tight against the laryngeal surface of the thyroid cartilage and not permitted any play; otherwise the pressures and suctions to which it was subjected during the acts of respiration, deglutition and coughing would soon have dislodged it from its anchorage, and thus it would have become a possible menace to the patient. Nor was it so tightly drawn that it might injure the tissues. The plate was of such shape that while resting in the larynx it did not interfere with the normal processes of breathing, coughing and swallow-

ing, and the sides were perfectly smooth and carried a high polish. The plate was of light material.

Though the plate may be left in place two weeks or longer without damaging tissues, in our cases it was removed ten days after introduction. While it was in place the patient could be ambulant in the hospital and could take the normally allotted amount of soft food without trouble. As a precautionary measure he was given penicillin.

The patient was seen and presented to the members of the Post-Graduate Otolaryngological Association three months after operation. At that time, three months after operation, the larynx was normal in every way. No vestige of a previous web could be seen. The patient's voice was clear, loud and resonant and in every way normal. On the anterior portion of the neck there was only a small, almost invisible scar.

Inasmuch as McHugh and Loch [1] have published a comprehensive review of 133 cases, citing 169 articles found in the literature, it would be superfluous to cover the same ground at this time. At the same time they reported a case seen by them in Johns Hopkins Clinic, in which, however, no attempt was made at correction of the obstruction.

SUMMARY

The procedure for eradication of a congenital laryngeal web is simple. The period of convalescence is short. The instruments required are a laryngeal trocar with a sharp-pointed stylet, a silver wire and a polished light metal plate.

140 East Fifty-Fourth Street (Dr. Seager).
745 Fifth Avenue (Dr. Poe).

1. McHugh, H. E., and Loch, W. E.: Congenital Webs of Larynx, Laryngoscope **52**:43-65 (Jan.) 1942.

MORPHOLOGIC DEFORMITIES OF THE LOWER LATERAL CARTILAGES

JACOB DALEY, M.D.
NEW YORK

IN THE article "Introduction of an Artistic Point of View in Regard to Rhinoplastic Diagnosis," [1] great emphasis is placed on the postoperative appearance of the nasal tip. In another article, "The Role of Columellar and Septocolumellar Sutures in Rhinoplasty," [2] it is stated that about 90 per cent of all artistic errors are centered around the nasal tip, involving the columella, the nostrils, the septolabial angle and the philtrum. Since the lower lateral cartilages form the major

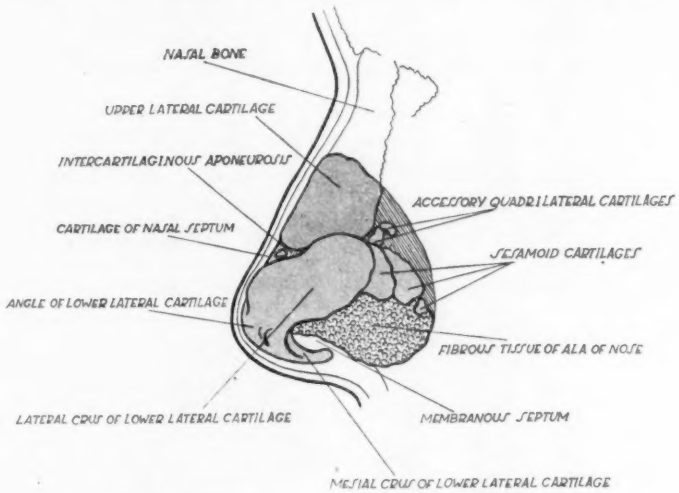


Fig. 1.—Anatomy of external nose (lateral view).

portion of the lower cartilaginous vault, it can readily be seen that they play an extremely important part in the appearance of the nasal tip. It naturally follows that one cannot consider a rhinoplasty adequate or

Read at the Fifth Annual Meeting of the American Otorhinologic Society for the advancement of Plastic and Reconstructive Surgery, Nov. 13, 1947, in New York.

1. Daley, J.: Introduction of an Artistic Point of View in Regard to Rhinoplastic Diagnosis, Arch. Otolaryng. **42**:33 (July) 1945.

2. Daley, J.: The Role of Columellar and Septocolumellar Sutures in Rhinoplasty, Arch. Otolaryng. **45**:178 (Feb.) 1947.

complete if the correction of deformities of these cartilages is neglected. To avoid modeling them is equivalent to retaining a bony hump when its removal is clearly indicated. This outstanding fault of many nasal reconstructions is due to the fact that many surgeons are reluctant to extend any surgical operation to the lower lateral cartilages.

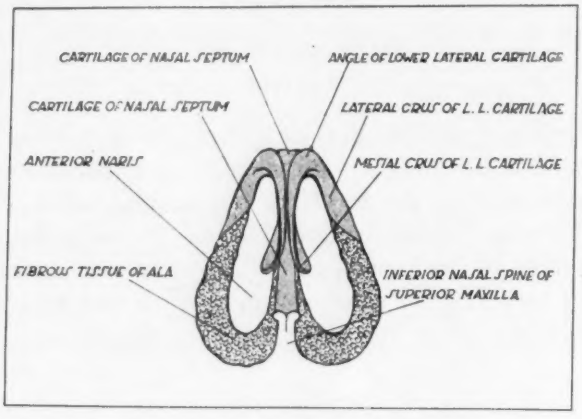

CARTILAGE OF NASAL SEPTUM
CARTILAGE OF NASAL SEPTUM
ANTERIOR NARIS
FIBROUS TISSUE OF ALA
ANGLE OF LOWER LATERAL CARTILAGE
LATERAL CRUS OF L. L. CARTILAGE
MESIAL CRUS OF L. L. CARTILAGE
INFERIOR NASAL SPINE OF SUPERIOR MAXILLA

Fig. 2.—Anatomy of external nose (basal view).

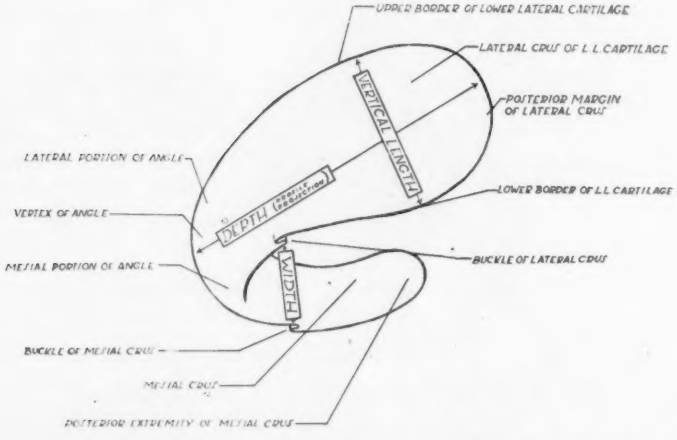


Fig. 3.—Schematic drawing illustrating the construction of the left lower lateral cartilage (see text).

It is fortunate that this attitude is gradually changing as more thought and attention are being focused on the correction of abnormalities and deformities of these cartilages. As soon as the surgeon acquires a clearer conception of their normal and of their abnormal anatomy and the exact procedure necessary for their surgical correction, he handles the lower cartilaginous vault with complete confidence.

ANATOMY OF THE LOWER LATERAL CARTILAGES (FIGS. 1, 2 AND 3)

The detailed anatomy of the lower lateral cartilages has been neglected by the anatomists, and few precise descriptions are available. These cartilages form the greater part of the lower cartilaginous vault and encircle the anterior part of the nostrils to maintain their patency. Each cartilage consists of a lateral and a mesial crura. These crura are continuous with each other, forming a rounded angle at the apex

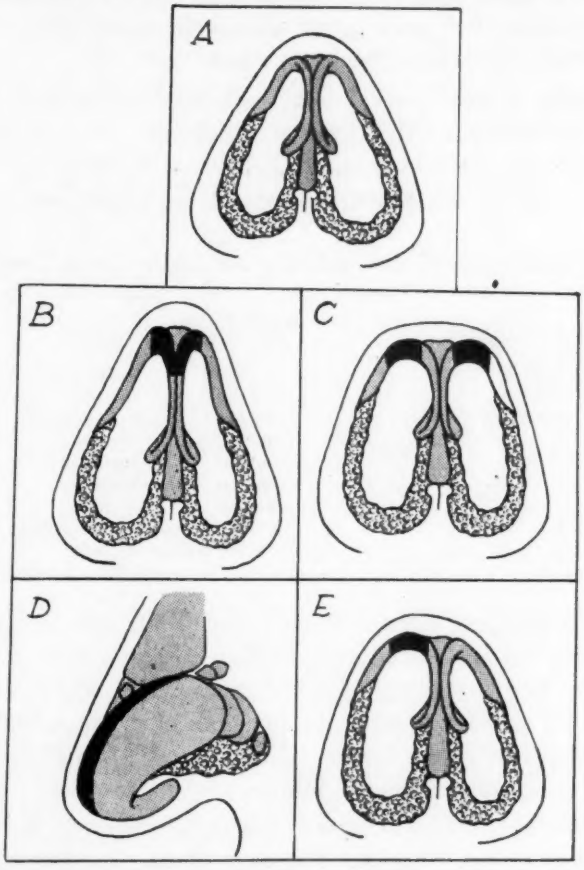


Fig. 4.—Basic types of morphologic deformities of the lower lateral cartilages and surgical correction: *A*, normal tip (basal view); *B*, prominent tip (basal view); *C*, broad tip (basal view); *D*, dependent tip (lateral view); *E*, asymmetric tip (basal view). The shaded areas indicate surgical correction.

of the nose. The arch formed by the lower lateral cartilage is U shaped, but instead of being on an even plane, it is sprung so that the mesial crus is at a lower level than the lateral crus (figs. 1 to 3).

The lateral crura are oval shaped and attached by their upper borders to the upper lateral cartilages by aponeuroses. The upper borders of

the lower lateral cartilages overlap the upper lateral cartilages. The connecting intercartilaginous aponeuroses form two intravestibular troughs called the limines vestibuli. Anteriorly the lateral crura blend into the angles, and posteriorly the borders are embedded in dense fibrous connective tissue which is attached to the maxilla (fig. 1).

The two mesial crura approximate in the mesial plane, and are connected above to the septal cartilage by the membranous septum. Anteriorly the mesial crura continue into the angles and the posterior ends extend about half way in the columella, curve slightly laterally and end in rounded extremities (figs. 1 and 2).

The angles of the lower lateral cartilages are arched and to a greater extent determine the shape of the lobule. At the apex of the nose they are separated by a notch and bound together by connective tissue. The septum lies above and behind the angles and is attached

TABLE 1.—*Morphologic Deformities of the Lower Lateral Cartilages*

Type of Nasal Tip	Depth (Profile Projection)	Width	Vertical Length
A. Prominent, broad, dependent	Increased	Increased	Increased
B. Prominent, broad	Increased	Increased	Normal
C. Prominent, dependent	Increased	Normal	Increased
D. Prominent	Increased	Normal	Normal
E. Broad, dependent	Normal	Increased	Increased
F. Broad	Normal	Increased	Normal
G. Dependent	Normal	Normal	Increased
H. Asymmetric	Due to difference in size or shape of the two lower lateral cartilages		

to them by connective tissue. Each angle blends into the lateral crus laterally and into the mesial crus mesially. The vertex of the angle differentiates into lateral and mesial portions. Frequently, slight twists or folds of the cartilage occur at the junctions of the angle and the crura and are designated as the buckle of the lateral crus and the buckle of the mesial crus, respectively (fig. 3).

The lower borders of the lower lateral cartilages do not extend to the free edges of the nostrils. The rims of the nostrils, being devoid of cartilage, are composed of fatty tissues, connective tissue and skin.

The lower lateral cartilages have three dimensions: depth (profile projection), width and vertical length. The depth of the cartilage is the distance from the vertex of the angle to the most distant point on the posterior margin of the lateral crus. The width is the greatest horizontal distance between the lateral and the mesial portion of the angle. The vertical length is the distance between the upper and the lower border of the cartilage (fig. 3).

The lesser alar cartilages are small and of a variable number. They are embedded in the fibrous tissue of the wings of the nose, between the lateral crus of the lower lateral cartilage and the maxilla. The sesamoid or accessory cartilages are usually from one to three in number and are located between the upper and lower lateral cartilages (fig. 1).

DEFORMITIES OF THE LOWER LATERAL CARTILAGES

The lower lateral cartilages may be deformed because of abnormalities of size, shape or position. In abnormalities due to size, one or more of their dimensions may be increased or decreased. Then again, in abnormalities of their shape or position, they may be twisted, buckled, overlapped, or displaced forward or downward by a long nasal septum.

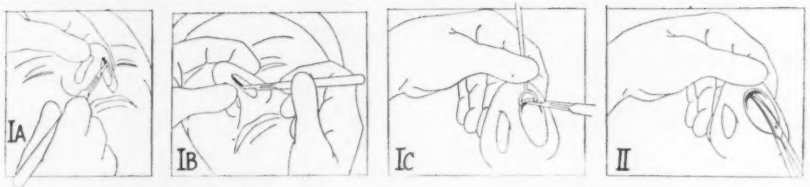


Fig. 5.—Surgical technic. Step I. Marginal Incision: *A*, incision of lower border of lateral crus. *B*, incision of lower border of mesial crus. *C*, incision of lower border of angle.

Step II. Separation of overlying tissues and skin.

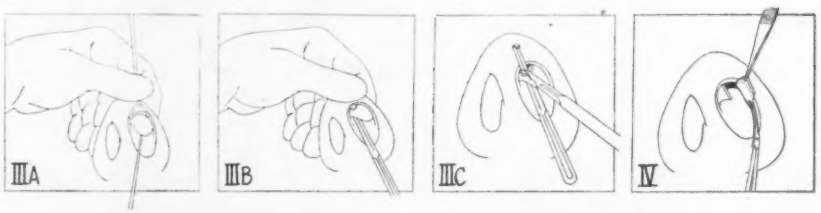


Fig. 6.—Surgical technic. Step III. Delivery of lower lateral cartilages: *A*, delivery and visualization of lower lateral cartilage. *B*, curved grooved probe in position. *C*, bisection of angle creating two cartilaginous flaps.

Step IV. Modeling of lower lateral cartilages: Removal of crescentic section of lower lateral cartilage.

These abnormalities are rather difficult to describe, but with a clear conception of the anatomic construction of the lower lateral cartilages, all deviations from the normal may be classified and each type subjected to the proper surgical correction. Any one or a combination of more than one of their components may participate in the deformity.

Since the variations and combinations of deformities are endless, it is not possible to give any set classification. Generally speaking, in morphologic deformities of the lower lateral cartilages, a prominent tip is due to an increase of the depth (profile projection), a broad tip

to an increase of the width and a dependent or hook tip to an increase of the vertical length (fig. 4). An arbitrary listing of the more common types of morphologic deformities is given in table 1.

SURGICAL TECHNIC

The technic used in the surgical correction of the lower lateral cartilages should be meticulous in every detail. The manipulation of these structures cannot be accomplished until the initial intercartilaginous incision and the transsection of the membranous septum have been made; therefore, the surgical alteration of these cartilages should be the final procedure in a complete rhinoplasty. If correction is limited to

TABE 2.—*Surgical Correction of Morphologic Deformities of Lower Lateral Cartilages*

Type of Nasal Tip	Surgical Correction
A. Prominent, broad, dependent.....	Reduce angles, mesial crura, lateral crura and upper borders of lateral crura
B. Prominent, broad.....	Reduce angles, mesial crura and as much of the lateral crura as necessary
C. Prominent, dependent.....	Reduce mesial portion of angles, mesial crura and upper borders of the lateral crura
D. Prominent.....	Reduce mesial portion of angles and mesial crura
E. Broad, dependent.....	Reduce angles, as much of lateral crura as necessary and upper borders of lateral crura
F. Broad.....	Reduce angles and as much of lateral crura as necessary
G. Dependent.....	Reduce upper borders of lateral crura
H. Asymmetric.....	Reduce the larger cartilage to match its mate

the lower lateral cartilages, the initial incision and transfixion of the nose must be made first.

The basic steps in this operation are (I) the marginal incision, (II) the complete separation of the overlying tissues, (III) the delivery and (IV) the modeling.

I. *Marginal Incision* (fig. 5).—The marginal incision is made inside the nostril through the vestibular skin and follows the lower border of the lower lateral cartilage. This lower border can be identified by a faint ridge just inside the nostril. The complete ridge is U shaped, faithfully following the course of the lower border of the cartilage.

Because of the flexibility of the nasal tip, the marginal or circumferential incision is a rather difficult procedure. It can be simplified, however, by pinching the nostril so that the columella is approximated on the collapsed alae. With a sharp no. 15 Bard-Parker blade, an incision is made running along and down to the lower border of the lateral crus from the anterior angle of the fold and extending posteriorly just short of the posterior margin of the lateral crus (*I A* in fig. 5).

The compressed tip is now turned in the opposite direction, and another incision is made along the lower margin of the mesial crus, extending from the anterior angle of the compressed tip, not quite to the posterior extremity of the mesial crus (fig. 5, *IB*). One now has two unconnected incisions exposing the lower margins of the lateral and mesial crura. A blunt double-pronged retractor is inserted in the angle of the nostril, and the nostril is everted. This brings the ridge corresponding to the lower margin of the angle into view. A third curved incision is now made along this ridge, connecting the two straight incisions so that the complete lower border of the lower lateral cartilage is exposed (fig. 5, *IC*). It is important that the marginal incision be continuous and uninterrupted by any bridges of vestibular skin. Extreme care must be taken to gage the proper depth of the incision in order to avoid cutting through the skin of the nasal tip.

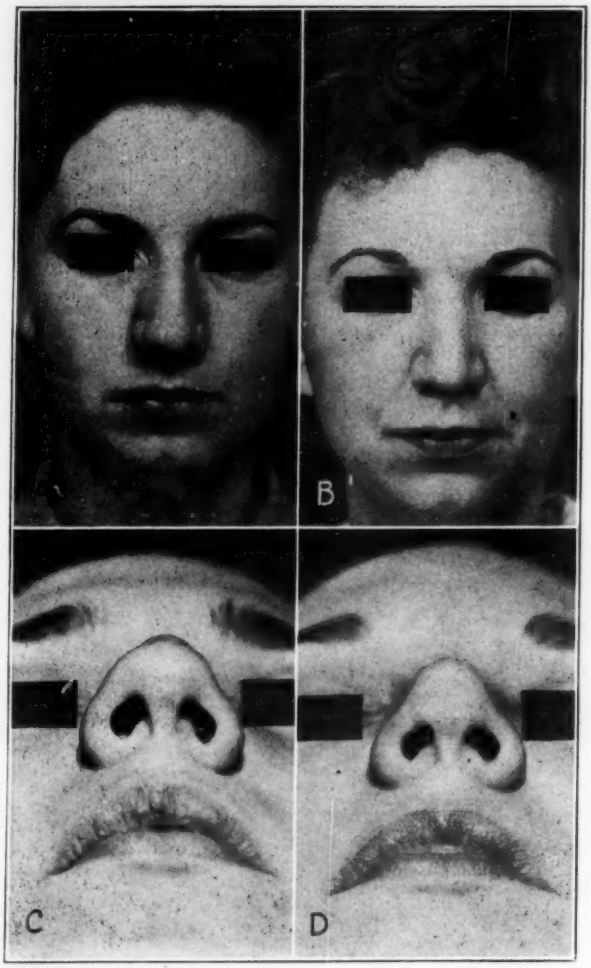


Fig. 7.—Photographs illustrating broad tip and correction: *A*, full face (before); *B*, full face (after); *C*, basal view (before); *D*, basal view (after).

II. *Separation of the Overlying Tissues and Skin* (fig. 5).—The next step in liberating the lower lateral cartilages is the careful separation of the overlying tissues. The blunt tip of a fine curved scissors is inserted point downward in the marginal incision at the angle. By separating the blades and taking care to stay in the plane above the perichondrium, the overlying tissues are freed (fig. 5, *II*). The separation must be carried high enough to extend beyond the initial intercartilaginous incision. When the point is reached, it is indicated by a sudden loss

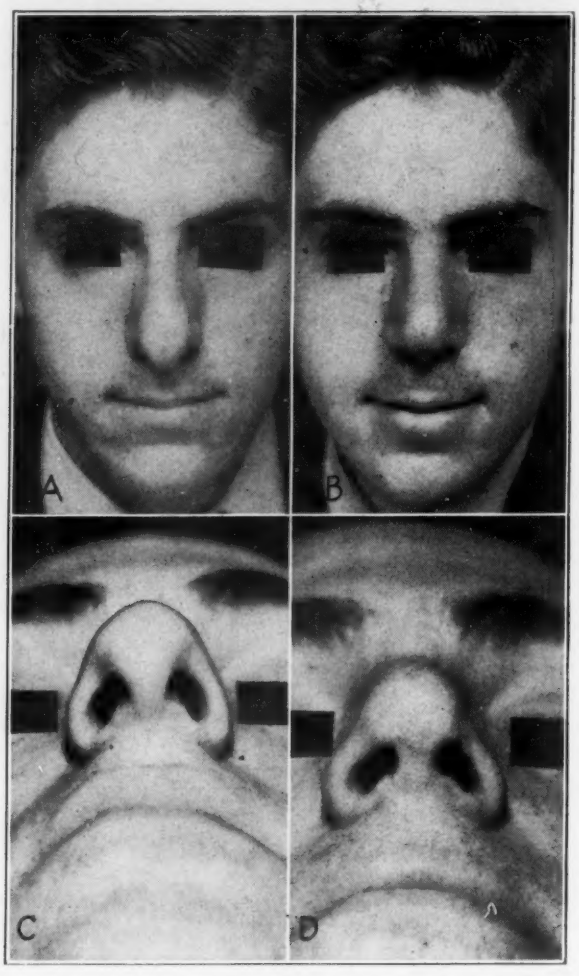


Fig. 8.—Photographs illustrating broad dependent tip and correction: *A*, full face (before); *B*, full face (after); *C*, basal view (before); *D*, basal view (after).

of resistance. The separation must also be continued laterally and mesially over the angle, so that the whole upper or superior surface of the lower lateral cartilage is detached from the skin of the nasal tip.

If the separation is complete, the next step becomes a simple matter. If, however, the separation is incomplete, little bands of tissue attached to the cartilages,

complicate the next procedure. Here again, extreme care must be taken not to fray the cartilages or traumatize the nasal skin, which is thin and delicate in this region of the nose (fig. 1).

III. *Delivery of the Lower Lateral Cartilages* (fig. 6).—Delivery of the cartilages is the next step. A double-pronged retractor is inserted in the marginal incision at the angle and the separated skin of the nostril is retracted. The surgeon

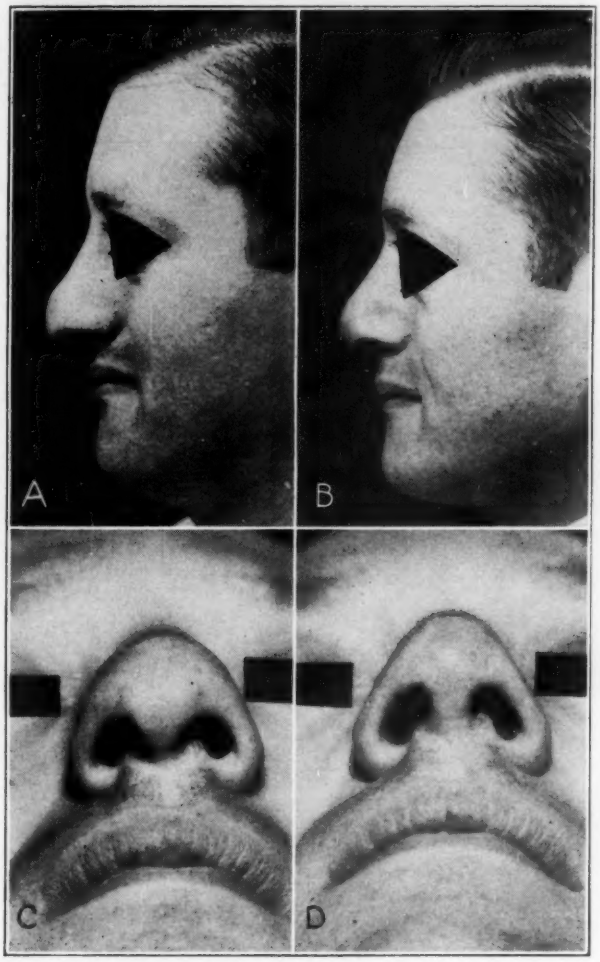


Fig. 9.—Photographs illustrating broad, dependent tip and correction: *A*, left profile (before); *B*, left profile (after); *C*, basal view (before); *D*, basal view (after).

pulls the cartilage down and out by means of a dural hook inserted in the vestibular skin at the angle. If the cartilage is completely freed, the downward displacement of the cartilage, accompanied by the upward retraction of the skin, will bring it into view (*III A* in fig. 6). The cartilage is now replaced in its bed and a Mackenty elevator or a curved, grooved probe is inserted inside the nostril and

pushed upward to the free upper border of the cartilage. By pulling the retracted skin up and over the tip of the instrument, the lower lateral cartilage is completely delivered and visualized (fig. 6, *III B*). With the instrument in place, the angle with the attached vestibular skin is bisected at its vertex with a no. 15 Bard-Parker blade. This bisection of the angle creates two cartilaginous flaps, which are now ready for modeling (fig. 6, *III C*).

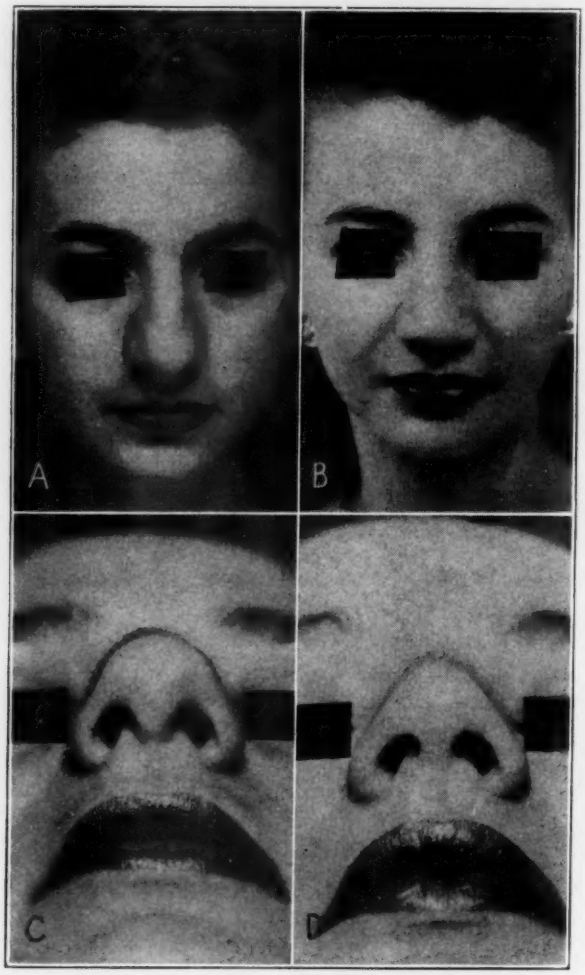


Fig. 10.—Photographs illustrating broad, dependent tip and correction: *A*, full face (before); *B*, full face (after); *C*, basal view (before); *D*, basal view (after).

An alternate method which I use is to split the angles without visualizing the lower lateral cartilages. This can be accomplished by the use of a small angular nasal speculum which resembles the Aufricht nasal speculum. This instrument is inserted in the marginal incision at the angle of the nose and is carried up beyond the initial intercartilaginous incision. The angle is bisected inside the nostril by cutting through the vestibular skin and the cartilage on the blade of

the speculum. This blade acts as a guard, preventing injury to the outer skin of the nasal tip. This technic is similar to the procedure used in separating the upper lateral cartilages from their septal attachments. This method prevents distortion and places the incision precisely at the vertex of the angle of the cartilage. When wide separation of the lower lateral cartilages is not necessary, this method is the one of choice.

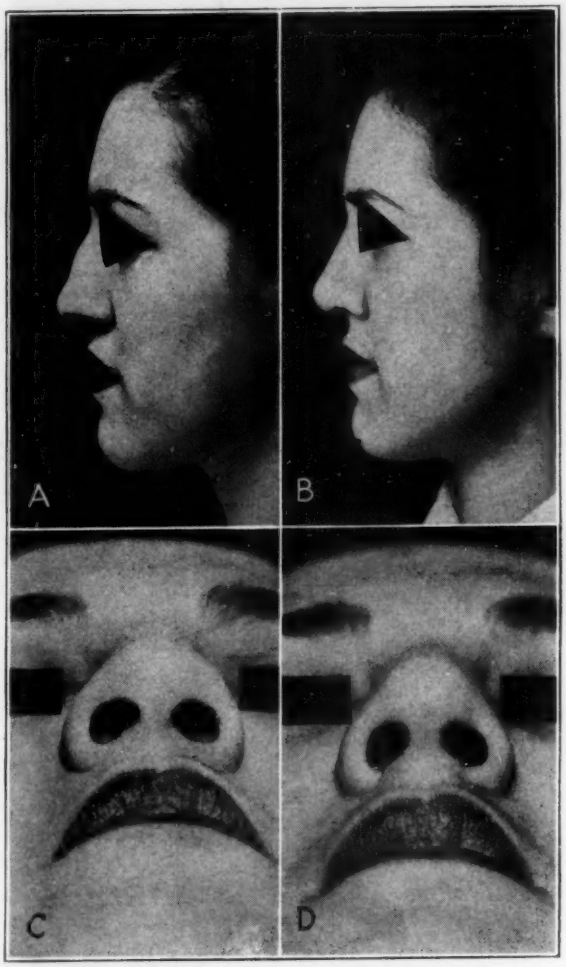


Fig. 11.—Photographs illustrating broad tip and correction: *A*, left profile (before); *B*, left profile (after); *C*, basal view (before); *D*, basal view (after).

IV. *Modeling of Lower Lateral Cartilages* (fig. 6).—Proper modeling of morphologic deformities of the lower lateral cartilages depends on observance of the following three rules:

1. To decrease the depth (profile projection), reduce the mesial portions of the angles and the mesial crura.
2. To decrease the width, sections must be removed from the mesial and lateral portions of the angles and as much of the lateral crura as is necessary.

3. To decrease the vertical length, the cartilages must be resected at the upper borders.

Reduction of the lower lateral cartilages is permissible in any area except along the lower borders in an anteroposterior direction. In reducing the vertical length, the excess should always be removed from the upper borders. Reduction of the lower borders will in this instance distort or destroy the clean lines and graceful

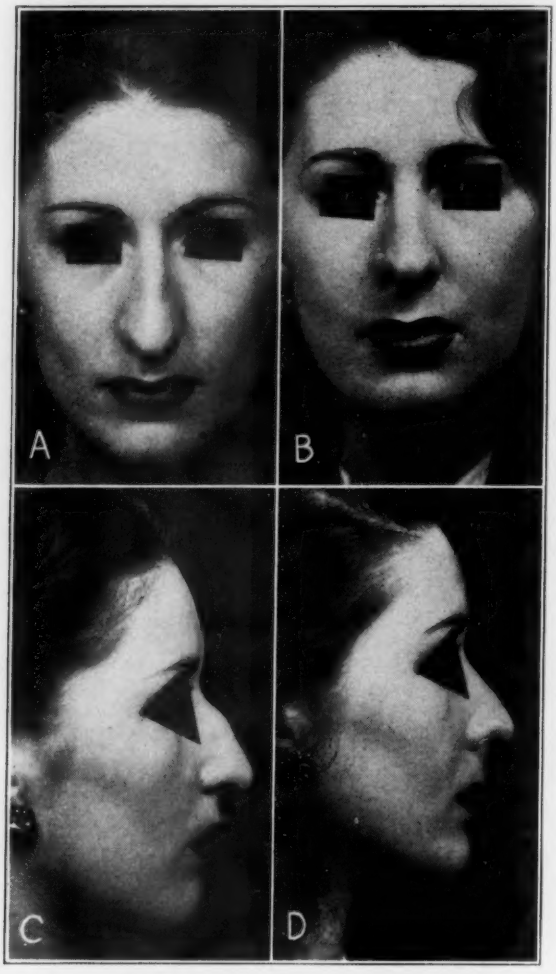


Fig. 12.—Photographs illustrating dependent tip and correction: *A*, full face (before); *B*, full face (after); *C*, right profile (before); *D*, right profile (after).

curves of the nostrils and substitute one deformity for another. The removal of cartilage in varying amount and from different areas will produce different effects. Hence the exact procedure necessary for the correction depends on the particular deformity involved.

It is important to keep in mind that although the cartilages are reduced in size, their original shape must be maintained. Therefore the angles and the crura are

reduced by removing curved sections. In reducing the angle and the upper border of the lateral crus simultaneously a crescentic section of the cartilage is removed. To help retain the original shape of the cartilage, the flaps are trimmed so that their vertical lengths match. All sharp corners are rounded off and the modeled structures are replaced in their normal positions.

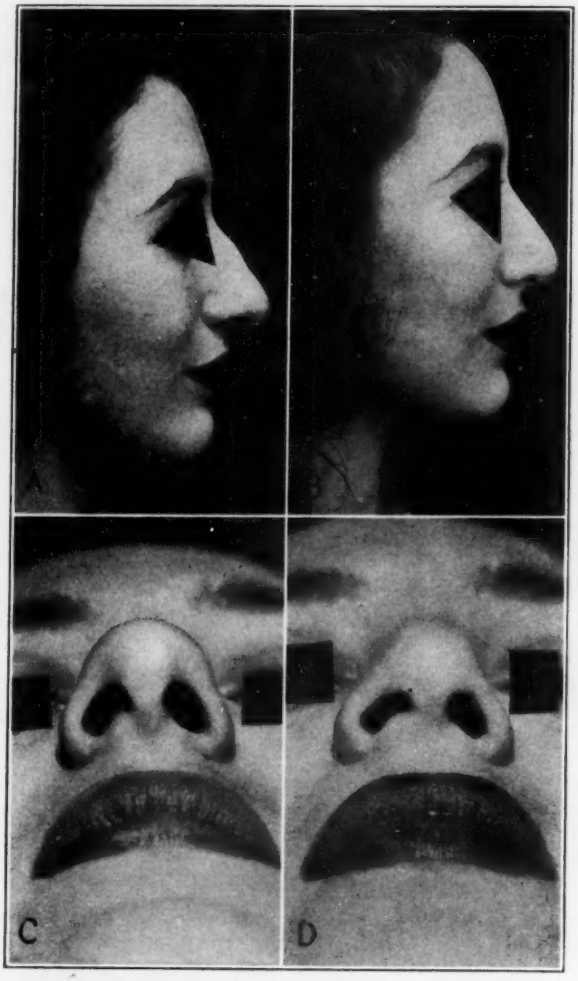


Fig. 13.—Photographs illustrating broad, projecting tip and correction: *A*, right profile (before); *B*, right profile (after); *C*, basal view (before); *D*, basal view (after).

Modeling the cartilaginous flaps is facilitated by grasping the vestibular skin with a dural hook. The flaps are steadied with a fine tissue forceps and are carefully trimmed with a pair of curved scissors.

The drawing illustrates diagrammatically the areas of the lower lateral cartilages to be reduced in different deformities (fig. 4). Reduction must be continued until the desired effect is attained, and every attempt should be made to match both sides to obtain symmetry.

Ordinarily, in resecting the lower lateral cartilages the vestibular skin is removed with the excess cartilage. This procedure reduces the size of the nostrils. On occasion these deformities are accompanied by small nostrils. This calls for a slightly different procedure. In this instance the vestibular skin must be separated from the under surface of the cartilages and preserved to prevent further reduction of the size of the nostrils.

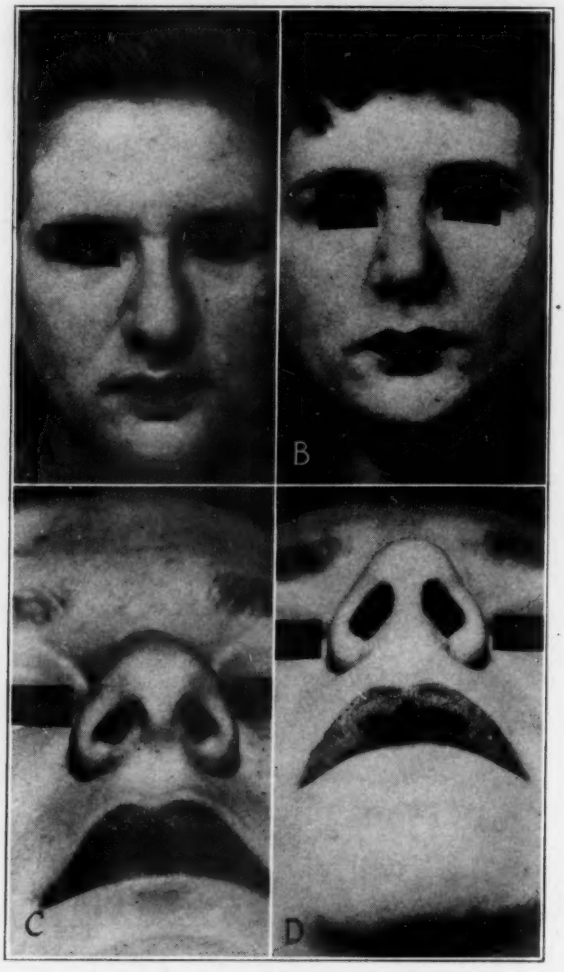


Fig. 14.—Photographs illustrating broad, asymmetric tip and correction: *A*, full face (before); *B*, full face (after); *C*, basal view (before); *D*, basal view (after).

In order to clarify further the surgical treatment of morphologic deformities, I have arranged another table outlining the proper surgical management of each type. Table 2 outlines the type of disfigurement and the appropriate surgical correction.

Since this article deals primarily with deformities of the lower lateral cartilages due to increase in size, the more common deformities not in this category are briefly discussed. They are as follows:

1. Flat or retracted tip, due to decreased depth in the anteroposterior direction.
2. Narrow tip, due to excessively narrow angles.
3. Retroussé tip, due to decreased vertical length with upward tilt.
4. Twisted tip, due to buckling or overlapping of the angles.
5. Cleft tip, due to separation of the angles.

When the cartilages are deficient in size as with a flat or a narrow tip, the dimensions may be increased by implantation of cartilage to make up the deficiency.[3] Depending on circumstances, living auricular or septal cartilage (autograft) or preserved cartilage (isograft) is used. A twisted tip may be corrected by complete separation of the lower lateral cartilages to permit them to return to their natural position. A cleft tip is corrected by separating the cartilages and approximating both angles with sutures.[3]

I do not pretend that this article covers the whole subject completely. Since deformities of the lower cartilaginous vault include those of the columella, alae and septolabial angle, it is impossible to cover this problem in one paper. Several phases of these disfigurements were discussed in recent articles,[4] and no doubt much more will be written on this subject as more thought is given to this fascinating problem.

Photographs illustrating some common types of hypertrophic deformities and their correction are included (figs. 7 to 14).

SUMMARY

It is essential to become thoroughly familiar with the normal and the abnormal anatomy of the lower lateral cartilages.

No rhinoplasty is complete without the correction of abnormal or deformed lower lateral cartilages.

Surgical technics and management of the lower lateral cartilages are explained.

A simple classification of the common types of morphologic deformities of the lower lateral cartilages and the methods of correction are outlined.

The drawings used in illustrating this article were made by Jeanne Verner.

104 East Fortieth Street.

3. Lamont, E. S.: Reconstructive Surgery of the Nose in Congenital Deformity, Injury and Disease, Am. J. Surg. **65**:17 (July) 1944.

4. Gatewood, W. L.: Correction of Multiple Deformities of the Nose, Virginia M. Monthly **71**:227 (May) 1944. Aufricht, G.: A Few Hints and Surgical Details in Rhinoplasty, Laryngoscope **53**:317 (May) 1943.

Case Reports

CALCIFIED HEMATOMA OF THE OROPHARYNX SECONDARY TO A GUNSHOT WOUND

EDWARD H. DIAMOND, M.D.
AND
CHARLES W. PERKINS, M.D.
NORWALK, CONN.

THE CASE herein reported was considered to be of interest because of the unusual nature of the condition and the absence of any report of a similar case in the literature. It is presented as a contribution to the differential diagnosis of pharyngeal tumors.

REPORT OF A CASE

A 70 year old retired policeman first sought medical advice, in August 1946, regarding progressive impairment of the hearing of the left ear. He stated that there was tinnitus in the left ear occasionally, and intermittent supraorbital pain

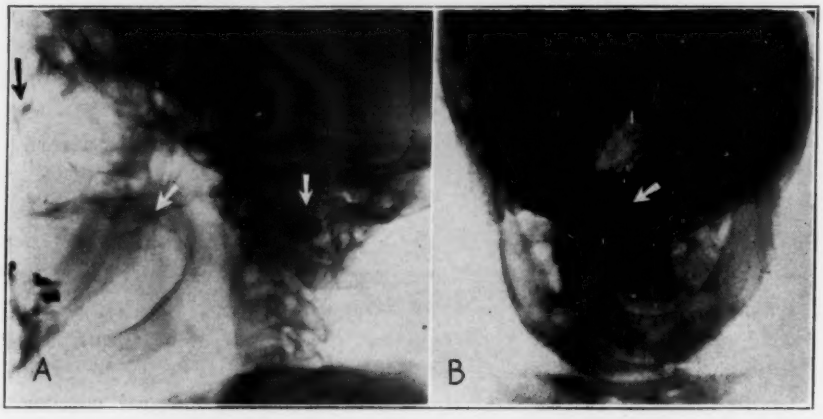


Fig. 1.—Roentgenograms taken in 1934, seventeen years after the injury. In *A* the arrows indicate the lateral passage of the bullet, which entered the left side of the nose and passed through the roof of the mouth to the atloaxoid region. *B*, anterior-posterior view of the lodged bullet.

on the left side. The past history disclosed that the patient had been shot in 1917 during the performance of duty, the bullet traversing the left malar region and lodging near the body of the second cervical vertebra. Following the injury, the patient was semiconscious for a time and had what apparently was partial left hemiplegia. At that time the patient was under the care of Dr. W. J. Tracey, of Norwalk, Conn., who subsequently referred him to the New York Post-Graduate Medical School and Hospital. The bullet was left in situ, and since that time there have been no symptoms except some soreness of the left parietal region and occasional pain in the left ear.

On examination the tympanic membranes presented a normal appearance. Hearing (whispered voice) was 15/15 in both ears. The sound of the tuning-fork was not lateralized in the Weber test. The Rinne phenomenon was positive in both ears. The left eustachian tube did not inflate readily. The nasal septum was moderately deviated bilaterally. In the throat the left lateral posterior pharyn-

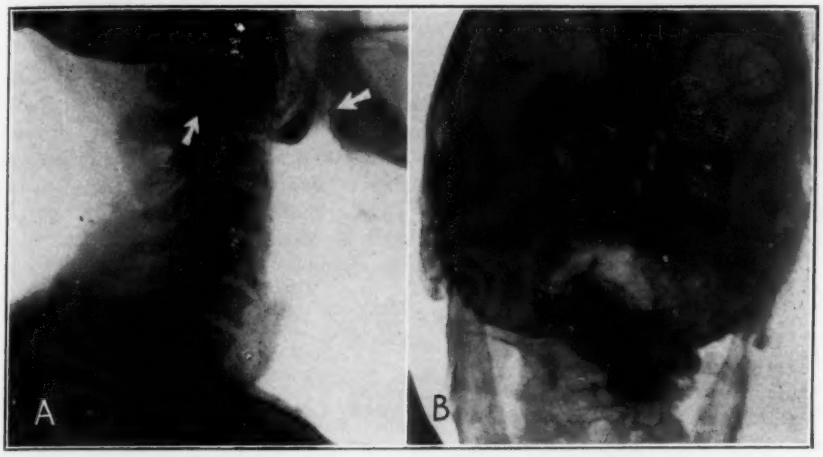


Fig. 2.—Roentgenograms taken in September 1946. *A* shows a calcareous deposit in the left side of the pharynx and vertebral region, obscuring the bullet. *B*, anterior-posterior view.

Fig. 3.—*A*, lateral view after aspiration of the area suggesting a neoplasm. *B*, anterior-posterior view after aspiration.

geal wall presented a large hemispheric swelling that was smooth and bony hard. This oropharyngeal mass was about 4 cm. in diameter.

The patient was referred to Dr. Charles W. Perkins for roentgenographic examination with a view to comparing the roentgenograms with some taken by

him in 1934. Unfortunately, those taken in 1917, at the time of the injury, were not available.

In figure *1A* and *B* are the films taken in 1934, which show that the bullet passed from the left side of the nose through the roof of the mouth to the atlas-axis region. In figure *2A* and *B* are the films taken in September 1946, showing a large calcareous deposit in the left side of the pharynx and vertebral region, obscuring the bullet.

As there was some question of a neoplasm, the patient was referred to the Memorial Hospital for the Treatment of Cancer and Allied Diseases, New York, for an opinion as to diagnosis and treatment. There the tumor was aspirated and 5 cc. of jelly-like material was obtained. The mass seemed diminished in size following the aspiration, but roentgenograms taken subsequent to this procedure (fig. 3 *A* and *B*) showed no change in the appearance of the mass. The pathologic report on the aspirated substance was: "calcific material."

It was agreed by all concerned that the mass was a calcified hematoma, not a true neoplasm, and that unless more serious symptoms developed, no further treatment would be indicated.

15 Belden Avenue.

520 West Avenue.

FIBROMA OF THE TRACHEA

JOHN S. KNIGHT, M.D., M.Sc. (Med.)
AND
WILLISTON P. BUNTING, M.D.
KANSAS CITY, MO.

FIBROMA of the trachea is of great rarity. Stangely enough, however, the first tracheal tumor of any type to be recorded was a fibroma and was observed as an incidental finding at an autopsy by Lieutad [1] in 1767. Since that time only 35 additional tumors of this type have been reported. The table presents a chronologic recapitulation of the cases and lists the authors reviewing them.

Offering further evidence of the extreme rareness of these tumors, Culp [2] completed the most recent exhaustive survey of primary tracheal tumors of all types in 1936 and listed at that time a total of 433. His figures, which were obtained from the contemporary literature and from a personal survey of the statistics of 47,600 autopsies, increased by only 3 the number of tracheal fibromas reported since the comprehensive report of D'Aunoy and Zoeller [3] in 1931.

REPORT OF A CASE

Mr. H. M., white, aged 20, a student of the University of Missouri, was first seen on Sept. 26, 1946, in Columbia, Mo., by Dr. C. R. Bruner and Dr. David LeMone, who deserve full credit for finding the tracheal tumor. The patient's chief complaints were bloody sputum, dyspnea and wheezing. He had reported for preschool football training and was a member of the "varsity" squad. Three weeks previously, he stated, he had coughed up a couple of mouthfuls of blood. However, strenuous exertion was accompanied by only mild dyspnea and slight wheezing. The past history indicated that he had had some blood-streaked sputum about a year previously, shortly after being released from the Army. In the spring of 1946 he had been a member of the track squad and had run the 440 yard dash without marked respiratory difficulty. No bleeding ever occurred during any sports competition.

---

1. Lieutad: Historia an. med., 1767, book IV, Obs. 64.

2. Culp, O. S.: Primary Carcinoma of the Trachea, J. Thoracic Surg. **7**:471, 1938.

3. D'Aunoy, R., and Zoeller, A.: Primary Tumors of the Trachea: Report of a Case and Review of the Literature, Arch. Path. **11**:589 (April) 1931.

Physical examination gave essentially negative results, the patient being a well developed, well nourished young man in no acute respiratory distress. Examination of the chest disclosed no abnormality.

A lateral cervical roentgenogram revealed a large mass protruding from the posterior wall of the trachea, 2 inches (5 cm.) below the larynx. There appeared to be almost complete occlusion of the tracheal lumen. Adenoma of the trachea was considered to be the most likely diagnosis.

Bronchoscopic visualization of the tumor showed a large lobulated reddish mass with intact mucosa about 2 inches below the vocal cords. The basal attachment to the posterior tracheal wall was firm and at least a half inch in diameter. The mucosa was continuous between the tumor and the tracheal wall. The decision

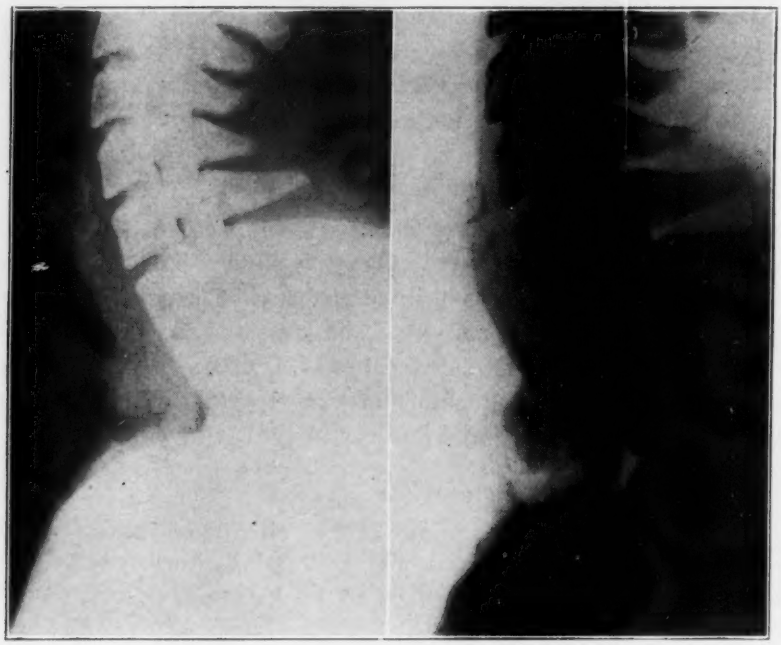


Fig. 1.—Negative and positive roentgenograms showing the location of the tumor.

was made to remove the tumor by external incision because of the extensive size, the broad base and the danger of hemorrhage. Local anesthesia was induced, and, with the head well extended, tracheostomy was performed through the fourth, fifth and sixth tracheal rings. There was no evidence of free pedunculation, and approximately four fifths of the airway was occluded. A mucosal incision was made at the junction of the tumor and the tracheal wall to find a sharply demarcated capsule outline which could be followed by dull dissection and clean enucleation. The party wall then appeared intact, and no excessive bleeding was present. Electrocoagulation was used to complete hemostasis, and the tracheostomy wound was closed tightly. Convalescence was uneventful, and the patient has returned to college and to his athletic endeavors.

The pathologic report[4] was as follows: "The material consists of a nodule measuring 2 cm. in diameter, which is lobulated, encapsulated, firm and friable. On section the lobules are separated by thin bands of dense fibrous tissue. Each lobule has a small lumen with papilliform tissue, and each lumen widens into a common central space. Histologically, the section is made up of spindle form and

Reported Cases of Primary Fibroma of the Trachea

Author	Date	Total Number of Cases as of That Date
Von Bruns: Neubildungen in der Luftrohre, in von Heymann, P.: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1898, vol. 1, chap. 2, p. 952	1898	23
Krieg, E.: Ueber die primären Tumoren der Trachea, Beitr. z. klin. Chir. **58**: 162, 1908	1908	25
Lombard, E., and Baldenweck, L.: Sur un cas de fibro-adénome de la trachée, Ann. d. mal. de l'oreille, du larynx **40**: 491, 1914	1914	29
D'Aunoy and Zoeller[3]	1929	33
Culp[2]	1937	36
Knight, J. S., and Bunting, W. P.: Arch. Path., this issue	1947	37

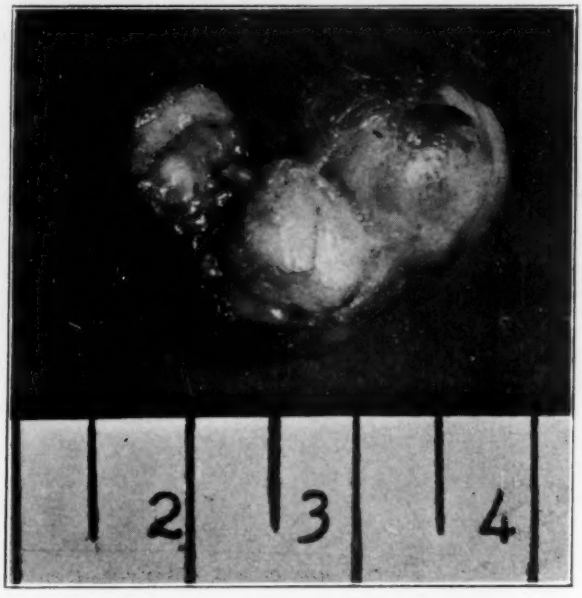


Fig. 2.—The tracheal fibroma removed.

oval cells. The nuclei vary, but moderately. Some edema is seen. Many of the cells contain abundant cytoplasmic prolongation. Some areas of degeneration are encountered, which are almost anuclear. Diagnosis: soft fibroma of the trachea."

4. The pathologic study was made by Ferdinand C. Helwig, M.D., St. Luke's Hospital, Kansas City, Mo.

Statistically, fibroma is the third most common benign tracheal tumor, being superseded by papilloma, then by the chondroma, osteoma and tracheopathia osteoplastica group, and followed closely by intratracheal goiter.

The tumor herewith reported is well within the succinct description of tracheal fibroma given by D'Aunoy and Zoeller,[3] which is quoted directly:

> The histologic changes present in the reviewed cases varied from nodular growths to polypoid tumors, pedunculated or broad-based, and soft or firm. Small cell infiltration varied considerably, as did vascularity. Usually, the tumors were covered with epithelium, varying in thickness in different areas and at times showing cornification. Hyaline degeneration was variable in amount, although generally present.
>
> From analysis of our statistics, as well as those collected by previous reviewers, it is evident that fibromas of the trachea occur at all ages, although they appear predominant in middle life, only three cases having been observed between the fifth and the fourteenth years. Sex incidence of tracheal fibromas is about equally divided. They have occurred in almost equal numbers in the upper and lower parts of the trachea. They have been found attached to the anterior and posterior walls about the same number of times.

1103 Grand Avenue.

Clinical Notes; New Instruments and Technics

A SUGGESTION FOR PACKING THE NOSE FOR ANESTHESIA

MAJOR J. JACOB
INDIAN ARMY, MEDICAL CORPS

PLACE the patient in exactly the same position as for tonsillectomy under general anesthesia, with a sandbag between the shoulder blades and the neck fully extended. In this position the roof of the nose, from which the nerves come, is in the most dependent portion of the nose.

Soak a 2 inch (5 cm.) length of ¼ inch (6.5 mm.) ribbon gauze in a 5 per cent solution of cocaine hydrochloride containing epinephrine hydrochloride and squeeze out the solution well. Place this along the floor of the nose with dressing forceps. Lift it up well against the floor. There should be no difficulty in cases of polypi. If there is a bad deflection, a curved probe will help.

A pea-size blob of cotton wool dripping with the same solution is placed in the anterior naris. The nostril is gently squeezed at intervals of three to five minutes.

Cocaine solution from the cotton wool goes along the ribbon gauze and trickles down all along the septum and the lateral wall of the nostril to the roof, where it remains if there is any excess. The nose is completely anesthetized in fifteen minutes.

The advantages of this procedure are:

1. No light is necessary. This work can be done blindly.

2. Excess of cocaine solution, if any, remains at the roof of the nose, doing useful work instead of being swallowed.

3. The greater the obstruction in the nostril (e.g., multiple polypi or a bad deflection) the better the capillary action and the less the quantity of solution needed.

(I have not seen this technic used by anybody, nor have I read about it anywhere. I have found this method of packing the nose for anesthesia most convenient and successful.)

% Dr. A. N. Verghese (Ret'd Civil Surgeon), Trichur, Cochin State, South India.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

THE PARANASAL SINUSES

SAMUEL SALINGER, M.D.
CHICAGO

THE LITERATURE of 1946 reveals the following trends: (1) less emphasis on surgical procedures, (2) increasing interest in the role of allergy and (3) widespread use of the antibiotics.

It is apparent that rhinologists are becoming more interested in the patient as a whole, widening their horizon beyond the narrow field of the nose and its accessory cavities. This is a healthy sign and should do much to overcome the ill repute into which the specialty had fallen by reason of the indiscriminate and widespread practice of surgery which was observable in certain quarters.

ANATOMY, PHYSIOLOGY, PATHOLOGY AND BACTERIOLOGY

Anatomy.—Giuseppe [1] presents an extensive study of metopism (persistence of the median frontal suture), based on roentgenologic examination in numerous cases as well as on examination of a number of skulls. Persistence of the fetal suture was found to be associated with agenesis of the frontal sinuses in the vast majority of cases. In the discussion of the pathogenesis the author cites numerous observers, setting forth a variety of possible etiologic factors, such as brachycephalia, hydrocephalus, hereditary tuberculosis, rickets, endocrine imbalance, craniofacial trauma, adenoids and craniorhinopharyngeal infections.

Marchi [2] studied the sphenoid sinuses in 100 skulls from the Anatomic Institute of Siena. They were sectioned in a frontal plane extending from a point just anterior to the tubercle of the sella turcica through the root of the pterygoid process. He found that in 84 per cent of the skulls the sinuses could be classed as large. Ostiums were classified as oval, round, semilunar and elongated. The incidence was as follows:

	Oval	Round	Semilunar	Elongated
Right	24	40	6	23
Left	15	42	4	35

1. Giuseppe, M.: Sutura metopica ipoplasia ed alterazioni morfologiche del seno frontale: Quadro anatomo-radiologico, Quaderni radiol. **7**:82, 1942.

2. Marchi, R.: Contributo allo studio del seno sfenoidale, Atti d. r. Accad. d. fisiocrit. in Siena **12**:40, 1944.

The author found that the ostium was usually high up and opposite the posterior end of the superior turbinate.

Smith [3] goes into great detail, describing measurements made in 120 skulls that were studied at the Benjamin Baptista Institute. He is particularly concerned with the "zygosphenoid" area or, more properly, the angle formed by the greater wing of the sphenoid bone and the lateral plate of the pterygoid process. Thirty-nine representative skulls are described in detail. No definite conclusions can be drawn, although the article should be of interest to anatomists.

Redoglia [4] discusses the various theories concerning the genesis of the frontal sinus and presents a case which he believes supports the theory that the sinus is developed from an anterior ethmoid cell. It had to do with a mucocele of the ethmoid sinus of a young woman, which was associated with aplasia of both frontal sinuses. He argues that the chronic inflammatory process which led to the formation of the mucocele exercised an inhibitory effect on the normal expansion of the ethmoid cells in the direction of the frontal sinus, thus halting the formation of the latter.

Physiology.—Proetz,[5] recalling that the normal ciliary activity provides a mechanism defending against infection, sets forth the agencies which may interfere with this mechanism and opens the way for infection resulting in the common cold. These are structural mechanical obstruction, drying, functional mechanical obstruction and biochemical stasis of the mucous blanket. The first three have been previously discussed and explained by the author and are pretty well understood. The fourth, however, involves the so-called "paralysis of the cilia," supposed to be due to virus invasion. The author suspects that this phenomenon may be analogous to an experiment shown by Prof. J. A. Harrison, in which certain antigen-antibody reactions in Paramecium produced an amorphous mass enveloping the cilia and preventing their propulsion. If experiments could be made on living mucosa duplicating the aforementioned experiment, they might go a long way toward enabling one to observe the behavior of the cilia under such conditions and might open the way toward solving the perplexing problem of the common cold.

Ruskin [6] analyzes the dynamics involved in vasodilatation and capillary permeability as seen in patients with allergic rhinitis and in

---

3. Smith, A.: Área zigo-esfenóidea, Arq. Inst. Benjamin Baptista **10**:171. 1944.

4. Redoglia, F.: Contributo alla conoscenga della embriogenesi del seno frontale, Boll. d. mal. d. orecchio, d. gola, d. naso **60**:3 (Jan.) 1942.

5. Proetz, A. W.: Nasal Physiology in Relation to the Common Cold, Ann. Otol., Rhin. & Laryng. **55**:306 (June) 1946.

6 Ruskin, S. L.: The Vascular Dynamics of the Common Cold and Allergic Rhinitis, Ann. Otol., Rhin. & Laryng. **55**:904 (Dec.) 1946.

patients susceptible to colds. He finds that these conditions will prevail if an imbalance exists between the vasoconstrictor epinephrine and the vasodilator acetylcholine. Experimentally and clinically, the administration of ephedrine has been found of value in covering and synergizing with the epinephrine, and capillary permeability will be resisted by a high calcium level and a hydrogen ion concentration above p_H 7. He therefore advises the administration of ascorbic acid, preferably as ephedrine ascorbate, and the calcium and sodium salts of ascorbic acid, for alkalinity.

Ornston [7] was able to demonstrate ciliary currents in the nose by insufflating some sulfathiazole microcrystals with an atomizer and observing the particles as they coursed down to the pharynx. The crystals seem to coalesce into small flecks, which are gathered together into streams that flow backward and merge in the nasopharynx. In the vestibule and on the anterior convexities of the turbinates, the particles become fixed by drying, since there they are directly exposed to the inspired air until sufficient mucus forms to wash them away. However, on the medial surfaces of the turbinates they move swiftly backward to the nasopharynx, flowing down the lateral walls. At times the streams may split over the torus palatinus. The direction of the streams is often affected by the presence of adenoid masses and of scars of previous surgical treatment. It was also found that septal deviations cause the particles to accumulate on a jutting spur, whereas on the concave side they move sluggishly, owing to the degeneration of the cilia that results from the slower interchange of air. Several colored photographs illustrate some of the points emphasized by the author.

Fabricant [8] reaffirms his conviction that a low p_H, indicating a slightly acid reaction of nasal secretion, represents a normal defense against infection, and offers additional data in support of his contention. Since vasomotor changes have been noted within the nose as a result of emotional states, leading to definite nasal symptoms, the author took occasion to measure the hydrogen ion concentration in several persons who either were shocked by sudden noise or were induced to exhibit excitement. Under these circumstances he observed a sudden rise in p_H, which persisted until the episode had passed off, when p_H again regained the normal value.

Pathology.—Keleman and Sargent [9] sectioned the heads of a number of apparently healthy rats and found definite pathologic changes in

7. Ornston, D. G.: Office Study of Cilia, Arch. Otolaryng. **44**:19 (July) 1946.

8. Fabricant, N. D.: Effect of Emotions on the Hydrogen Ion Concentration of Nasal Secretion in Situ, Arch. Otolaryng. **43**:402 (April) 1946.

9. Kelemen, G., and Sargent, F.: Nonexperimental Pathologic Nasal Findings in Laboratory Rats, Arch. Otolaryng. **44**:24 (July) 1946.

the nasal cavities in 60 per cent of them. Since these animals had not been subjected to any previous experimental tests and were presumed to be normal, it appears to the authors that deductions based on findings in so-called normal controls warrant great caution and possible revision.

Wright [10] presents an analysis of 75 cases in which a round shadow suggesting a cyst of the maxillary sinus was discovered by roentgen examination. In 48 cases surgery was resorted to, affording an opportunity to study the histologic aspects and to coordinate these with the clinical history. In 19 of the cases a nonsecreting cyst alone was found, without other changes in the mucosa. Two or three such cysts were present in 9 cases, and in 7 others such a cyst was disclosed in conjunction with a mucous cyst or a polypoid membrane. Other findings were a mucous or a secreting cyst alone in 3 cases, a polyp in 5 cases, atypical cysts in 2 and a polypoid membrane in 1. A wide variety of symptoms was encountered in many instances, having no direct connection with the pathologic appearance of the sinus.

In many instances there were no symptoms directly attributable to the cyst. Apparently, it is only when the cyst attains sufficient size to cause pressure that the patient will complain of pain, soreness or numbness in and about the cheek or the teeth. Indirectly, the cyst may give rise to headaches, vague pains, fatigue, low grade fever and other signs suggesting a low grade infection. Absorption of toxin has been noted by other authors. Several illustrations of tissue removed at operation disclose two typical varieties, the nonsecreting and the mucous cyst. The former seems to be the result of distention of the subepithelial areolar spaces, while the latter is caused by distention of a mucous gland.

Bacteriology.—Sparrevohn and Buch [11] cultured washings in 196 cases of maxillary sinusitis, obtained by puncture of a total of 272 specimens, and found that pneumococci predominate in the acute type whereas streptococci were more common in the chronic. Mixed infections were encountered mostly in the cases of chronic maxiliary sinusitis and usually after several irrigations; the authors ascribe them to introduction of secondary invaders. In a number of cases the cultures were sterile. This may have been due to death of the bacteria, occurring before the cultures reached the laboratory, or to subsidence of the infection. Clinically, the time required to cure the sinus was shorter when cultures were sterile, longer when pneumococci were present and longest when there was streptococcic infection. The percentage of patients coming to operation was practically the same in all three circumstances.

10. Wright, N. W.: Round Shadows in the Maxillary Sinus, Laryngoscope **56**:455 (Aug.) 1946.

11. Sparrevohn, U. R., and Buch, A.: The Bacteriology of Maxillary Sinusitis, Acta oto-laryng. **34**:425, 1946.

DIAGNOSIS

Mangabeira-Albernaz [12] reviews the various symptoms and findings in chronic sinusitis from the diagnostic point of view. Among other things he mentions the rates at which solutions are absorbed and eliminated from the maxillary antrum. A 5 per cent solution of azosulfamide is instilled into the antrum. Normally it should appear in the urine in thirty minutes and be entirely eliminated in twelve to sixteen hours. When the mucosa of the sinus is diseased, absorption is slower, and therefore the elimination of the drug is delayed as long as thirty-six hours.

Among the various roentgen projections, he stresses the value of that obtained with the Hirtz position, in which the ethmoid and sphenoid sinuses are shown between their natural boundaries. Comparison between the two sides is much easier in this view.

Also, in the matter of displacement he favors the procedure of Ermiro de Lima over the Proetz method.

Ibsen [13] states that basal convex shadows showing in roentgenograms of the maxillary sinuses may be caused by any of the following: surface exudate, localized acute sinusitis, mucocele, solitary mucous polyp, retention cyst, dental cyst or projection of contours from without. Analysis of 35 cases, of which 30 were subsequently reexamined, showed that in 18 of these the shadow had disappeared spontaneously. It would appear, therefore, that this finding cannot in itself be considered an indication for surgical intervention.

Eliseht [14] points out that antral polyps and dental cysts frequently present the same type of roentgenographic shadow. The differential diagnosis may be simplified by the use of a contrast medium, which will show the origin of the former to be in the region of the ostium, whereas the cyst grows upward from the alveolar region. He considers the diagnosis important from the therapeutic standpoint, because in the case of a cyst the surgical procedure must include removal of the offending tooth.

INTRACRANIAL COMPLICATIONS

Among 37 cases of meningitis seen by a clinical group [15] 9 were due to sinus infections and 7 to fractures extending through the

12. Mangabeira-Albernaz, P.: Estado atual do diagnóstico das sinusites crônicas, Brasil-med. **59**:255 (July 21-28) 1945.

13. Ibsen, B.: Basale konvekse kaebehulesloringer, Nord. med. **27**:1487 (July 25) 1945.

14. Eliseht, F. C.: Quistes y polipos del seno maxilar, Prensa méd. argent. **33**: 513 (March 8) 1946.

15. Smith, H. V.; Schiller, F., and Cairns, H.: Discussion on the Chemotherapy of Meningitis Secondary to Infection of the Ear and Nasal Sinusitis, Proc. Roy. Soc. Med. **39**:613 (Aug.) 1946.

sinuses. The bacterial invaders in the cases due to sinus infections were pneumococci in 6 cases and streptococci in 3; in the cases due to fractures, they were pneumococci in 6 and streptococci in 1.

Penicillin, 2,000 Oxford units to the cubic centimeter of solution, was used intrathecally in the cases of meningitis. Daily spinal injections of 8,000 to 10,000 units were given after withdrawal of about 6 cc. of fluid. The spinal injections were kept up for at least five days. Recurrences were frequent if the treatment was stopped too soon. At the same time sulfadiazine was given orally in doses of 12 Gm. daily. Also, penicillin was given either intravenously or intramuscularly in doses of 120,000 to 320,000 units in twenty-four hours. The authors point out that the intrathecal injections are not without danger. A nonirritating brand of penicillin must be selected, their choice being crystalline penicillin made by Glaxo, Ltd. Excessive doses (40,000 units or over), especially if concentrated, may produce severe cerebral reaction. If the solution or the procedure is not perfectly aseptic there is danger of introducing secondary infection. Likewise, there is danger in repeated spinal taps when the pressure is high, as in cases complicated by brain abscess.

The latter is treated by aspiration, followed by instillation of a more concentrated penicillin solution. The amount depends on the size of the abscess; usually it is 1 to 3 cc. If the abscess is large, aspiration alone will relieve the pressure. Otherwise, it will be necessary to do a ventricular or a spinal tap.

For the treatment of a subdural abscess, fine rubber catheters are inserted through suitable drill holes, numbering at least five.

Concerning surgical treatment, the authors feel that it may be delayed because of the favorable action of the antibiotics. However, when meningitis follows a fracture extending through the sinuses, it is imperative that the dural defect be repaired as soon as the meningitis is brought under control.

The results in the entire series were better in the cases of pneumococcic than in those of streptococcic infection, probably because of the higher percentage of the latter in which brain abscess occurs.

An extensive discussion followed, in which several of the men gave varying opinions as to the amount of penicillin to be used and as to the proper time for surgical intervention. The tendency of meningitis to recur after apparent cure was mentioned by the brain surgeons, who felt that an undrained focus was the cause of recurrence. It seemed to be the consensus that whereas penicillin held out hope in what were previously considered hopeless cases, it was still important that the brain surgeon be consulted in these serious cases.

Vincent, Thiebaut and Daum [16] report 3 cases of extradural abscess due to disease of the sphenoid and ethmoid sinuses and comment on the rarity of this complication. The diagnosis, which in these cases was made only on surgical intervention, is usually difficult. This complication can occur only if the anatomic configuration of the sinuses involved is of a certain type; i. e., the sphenoid sinus is prolonged into the lesser wing, or a posterior ethmoid cell encroaches on the optic nerve. The symptoms noted were pains in and about the eye, often hemicrania, unilateral impairment of vision without scotoma or fundus changes, diplopia due to oculomotor paresis, exophthalmos and symptoms of intracranial pressure, such as vomiting, subnormal temperature and increased pressure of cerebrospinal fluid. Roentgenograms will reveal involvement of the posterior sinuses and frequently some absorption of bone at the internal portion of the greater and lesser wings, as well as of the body of the sphenoid bone. A history of previous acute sinus infection may arouse suspicion; if this was combined with local pains and ocular symptoms, attention is drawn to the middle fossa of the skull. An intranasal examination is often misleading, since the primary sinus infection may have subsided. If one is mindful of the possibility of an intracranial infection, the likelihood of mistaking it for a meningioma or an aneurysm of the internal carotid artery will be reduced.

The authors advise that the surgeon operate in two stages: First, the neurosurgical, in which a frontal or a frontoparietal craniotomy is done and the frontal lobe elevated, exposing the sphenoethmoid region. Here, in 2 cases, they found a soft tumefaction pushing the dura outward, which on puncture yielded a quantity of thin dark pus. Frequently a fistula is found leading into the sinus. This is enlarged to permit the pus to drain freely into the nose. The dura is then closed, as is the cranium, and the second step is undertaken, which consists of an intranasal exenteration of the posterior sinuses. Two of their 3 patients recovered.

Pote and Courville [17] found intracranial complications due to disease of the nasal passages and sinuses in 160 of 30,000 consecutive autopsies. Since in 42 of the cases the intracranial involvement was associated with otitis media, it was difficult in this group to determine which focus of infection was the prime etiologic factor. The association of otitis media and sphenoiditis in the presence of meningitis is a well known complex that has been the subject of varying interpretations. These authors found that disease of the frontal sinus was responsible for

16. Vincent, C.; Thiebaut, F., and Daum, S.: Les abcès extra-duraux d'origine sphéno-ethmoïdale, Rev. de méd., Paris **60**:166 (July-Oct.) 1943.

17. Pote, W. W. H., and Courville, C. B.: Intracranial Complications of Infections of the Nasal Air Passages and Accessory Sinuses, Bull. Los Angeles Neurol. Soc. **10**:114 (Sept.-Dec.) 1945.

the greater number of the complications, and these included extracranial, cranial and intracranial infections of every possible form. Isolated ethmoiditis was rarely the cause of intracranial complications, and then only in cases in which the infection passed into the cribriform area or involved the cavernous sinus. Infection of the maxillary sinus alone seldom caused intracranial complications. In cases in which it did, the involvement was the result of a spread of osteomyelitis. Malignant tumors and malignant granulomas were responsible in a few cases. The report is an interesting one because of the wealth of material, the care with which it was studied, and the information gained as to pathways of infection.

Ebert [18] reports 2 cases of purulent meningitis complicating acute sinusitis in which recovery occurred without the aid of antibiotics. In one case streptococci were obtained from the spinal fluid; in the other there were no bacteria. However, in both cases, the symptoms were typical, and the spinal fluid, under great pressure, was cloudy and contained a large number of cells. The author in explaining the happy result states that the primary focus was eliminated through repeated irrigations of the antrums, which yielded a great deal of pus, and that frequent lumbar punctures were resorted to. He feels that while antibiotics may be of value, they cannot be depended on for a cure if the infected focus in the sinus is not adequately drained.

In a beautifully illustrated article, Robertson [19] gives the details of a case in which an abscess of the frontal lobe following an acute infection of the upper respiratory tract of a boy of 15 was successfully treated. The gradual obliteration of the large abscess cavity was observed by means of roentgenograms, gas being used as a contrast medium. When roentgenography was combined with encephalography, it was possible to follow the progress of the collapsing cavity as well as its relationship to the lateral ventricles.

Rushton's [20] patient was a 12 year old girl with a history of pyrexia, increasing lethargy and frontal headache of ten days' duration. There was some swelling as well as tenderness over the bridge of the nose, with opacity of the right antrum and both frontal sinuses. Puncture of the antrum yielded a thick foul greenish pus. A penicillin solution was instilled every three hours. The patient became more lethargic, the swelling increased, and there were other evidences of intracranial pressure. The frontal sinus was opened and found to contain thick

18. Ebert, E.: Deux cas d'inflammation des fossés nasalés et des cavités accessaires avec méningite purulente terminée par guérison, Progrès méd. **74**: 165 (April 24) 1946.

19. Robertson, E. G.: Intracranial Abscess Secondary to Frontal Sinusitis, Roy. Melbourne Hosp. Clin. Rep. **16**:60 (Dec.) 1945.

20. Rushton, P. C.: A Case of Subdural Abscess Secondary to Acute Frontal Sinusitis, J. Laryng. & Otol. **61**:217 (March) 1946.

yellow pus. A tiny erosion was found in the posterior wall, through which pus exuded. The posterior wall was removed, uncovering an extensive extradural abscess. The patient recovered after two weeks, during which penicillin and sulfadiazine were being administered.

Ferrannini's [21] patient was a woman aged 27 who had complained of severe headaches over a period of several months and recently of failing vision. There was a history of frequent nasal obstruction and discharge. A large choanal polyp obstructing one side was removed, and removal was followed by considerable relief of the headache. However, the vision continued to deteriorate, and examination revealed a marked degree of optic atrophy. Roentgenograms failed to reveal anything abnormal. Lumbar puncture yielded a clear fluid under slightly increased pressure, which showed an increase in the number of cells, mostly lymphocytes, and in albumin. The Wassermann test was negative. The diagnosis was circumscribed basilar serous meningitis secondary to chronic ethmoiditis. The patient was treated with mercury preparations and iodides without effect. The blindness became total and permanent.

The author devotes considerable space to a discussion of the pathogenesis of this condition, without being able to offer anything hopeful in the prognosis.

Weder [22] analyzes 6 cases of orbital and intracranial complications of sinusitis and is generally impressed with the effectiveness of penicillin in combination with a sulfonamide compound. He notes further that penicillin is particularly effective against all staphylococci, with the exception of certain strains of Staphylococcus albus; also, that penicillin is safe and may be easily applied locally and intrathecally. Furthermore, he points out that there is less danger from penicillin than from sulfonamide drugs in severely toxic patients. The author sets forth the following rules of procedure, which he believes have given him the best results: In all cases of severe suppurative sinusitis with high fever large doses of sulfonamide compounds are given in connection with the other usual local procedures. If within forty-eight hours definite improvement has not appeared or there are symptoms of beginning complications, penicillin is added and operative procedures are instituted to uncover and remove the focus. Penicillin solutions are used locally in all operational cavities.

Among 81 cases of meningitis due to diseases of the ear and the nose assembled by Opheim [23] were 20 in which the meningitis was rhinogenic.

---

21. Ferrannini, L.: Meningite basilare anteriore circonscritta e nevrite ottica retro-orbitaria propagate da etmoidite, Minerva med. **33**:453 (Nov. 17) 1942.

22. Weder, A.: Beitrag zur Sulfonamid—und Penicillin—anwendung bei akuten Nasennebenhöhlen-eiterungen und ihren lebenbedrohenden Komplikationen, Pract. oto-rhino-laryng. **8**:1, 1946.

23. Opheim, O.: Otogene og rhinogene meningitter: Kjemoterapi, Nord. med. **12**:3583 (Dec. 13) 1941.

In 9 of these chemotherapy was used, and this group combined with 31 cases of otogenic meningitis in which the treatment was similar yielded 15 recoveries. These cases are compared with 41 cases in which antibiotics were not employed and in which there were only 8 recoveries. The conclusion is obvious. Sulfapyridine seemed to act better than sulfanilamide. (The article, though interesting, is nevertheless outdated, having appeared in 1941. Many of the Scandinavian journals of the war years reached us only during the past year. S. S.)

Schultz [24] reports 2 cases of subdural abscess complicating infection of the frontal sinuses, one of which was fatal. The seriousness of this complication cannot be overemphasized, and the author stresses the importance of suspecting its presence in cases of sinusitis in which the symptoms cannot be explained. Jacksonian convulsions, hemiplegia or aphasia occurring in the presence of frontal sinusitis usually indicate subdural empyema. Drainage of the subdural space is accomplished through multiple burr holes and the liberal use of antibiotics.

A case of pneumococcic meningitis reported by Langeron and his associates [25] is interesting because of an unrecognized fracture which was obscured by an old osteoma and which resulted from a minor trauma. Large doses of sulfonamide compounds reaching a blood level of 20 mg. per hundred cubic centimeters succeeded only temporarily in arresting the process, which persisted for a month, with acute exacerbations, until penicillin was finally obtained and given both intramuscularly and intrathecally. The fracture was revealed at the time the osteoma was removed, several days after the penicillin therapy was instituted. The improvement was immediate, and complete recovery ensued.

Ebert [26] reports 2 cases of purulent meningitis associated with pansinusitis (roentgenograms not mentioned) in which recovery occurred under drainage alone. In 1 case the spinal fluid revealed streptococci. No mention is made of any antibiotics having been employed. The author stresses the importance of drainage. In both cases the nose was cleaned, the antrums were irrigated and several spinal punctures were performed. The average duration from onset to recovery was three weeks. The importance of examining and treating nasal infection in cases of meningitis is emphasized in a number of references to the literature.

---

24. Schultz, E. C.: Subdural Empyema of Frontal Sinus Origin, Ann. Otol., Rhin. & Laryng. **55**:882 (Dec. 1946.

25. Langeron, L., and others: Méningite à pneumocoques traumatique chez une malade porteuse d'un ostéome du sinus frontal: Plusiers rechutes; action des sulfamides, de la pénicilline et de l'exérèse de l'ostéome, Bull. et mém. Soc. méd. d. hôp. de Paris **62**:112, 1946.

26. Ebert, E.: Två fall av inflammation i näsans bihålor, purulent menengit, hälsa, Nord. med. **25**:542 (March 23) 1945.

Malis [27] presents the details of a case of frontal lobe abscess developing in a soldier a few weeks after acute frontal sinusitis which had left him with a draining fistula. Despite prolonged treatment with antibiotics the symptoms of intracranial pressure developed, which led to an operation disclosing an encapsulated abscess communicating with the sinus by way of a fistula in the dura. Following the operation, blindness developed on the affected side, also hemiplegia. The former was attributed to thrombosis of a central vascular radical and the latter to the adjoining encephalitis involving the cortex. The patient recovered after a stormy five weeks, during which time he received large doses of penicillin intramuscularly and intrathecally. The author points to drainage as a determining factor in the recovery, though crediting the antibiotics with having controlled the spread of the infection.

A case of frontal lobe abscess with recovery is reported by Thomas.[28] The patient was a youth of 16. There was no history of trauma. The onset was rapid, with headache, vomiting, fever and a fluctuant red swelling over both frontal sinuses. These were opened externally and contained some thick pus, which when cultured yielded Staphylococcus aureus and diphtheroids. Despite good drainage and sulfonamide therapy the boy became drowsy and semiconscious and manifested other signs of intracranial involvement. A wide exposure disclosed extensive necrosis of the frontal bones with sequestration. A sinus was found on the anterior aspect of the right frontal lobe, which yielded pus on probing. A drain was inserted and penicillin therapy instituted. Despite the patient's general improvement the sinuses continued to drain, and it was later necessary to open them both and clean out polypi and diseased membrane. After complete healing the roentgenograms showed a bony defect measuring 7.5 by 5 cm.

ORBITAL COMPLICATIONS

Cullom [29] is convinced that sinus disease may be responsible for various ocular disorders and cites 16 cases to support his contention. Included are 6 cases of intractable chronic ulceration of the cornea, 4 cases of toxic amblyopia, 2 cases of retinochoroiditis, a case of retinitis circinata and 2 other cases. In every instance definite evidence of sinus involvement was seen in the roentgenograms, and radical operation was followed by prompt and complete cure of the ocular

27. Malis, S.: Frontal Lobe Abscess, Eye, Ear, Nose & Throat Monthly **25**: 498 (Oct.) 1946.

28. Thomas, E. W.: Frontal Lobe Abscess Treated with Penicillin, Brit. M. J. **1**:12 (Jan. 5) 1946.

29. Cullom, M. M.: The Role of Sinusitis in Eye Pathology, Laryngoscope **56**: 164 (April) 1946.

disease. The author comments on the fact that these patients all exhibited symptoms of toxemia, such as loss of weight, pallor, lack of appetite, etc. The improvement in their general condition was as marked as the relief of their ocular symptoms.

Gallo and de Castro [30] discuss ocular and orbital complications of sinus disease, dividing them into two groups: (1) the manifest suppurative complications arising from the anterior ethmoid and frontal sinuses, about which there is a fair unanimity of opinion, and (2) the infections arising from the posterior sinuses, which cause the more serious complications of cavernous sinus thrombosis, orbital phlegmon and optic neuritis, and concerning which there is some difference of opinion with regard to the pathogenesis.

In connection with optic neuritis particularly, the author mentions the various theories under the headings "Infections" and "Mechanical Pressure," citing various authors. No particular conclusions are drawn.

Godtfredsen [31] assembled 50 cases of orbital complications following operations on the sinuses, including 8 of his own. The latter were observed during the period between 1939 and 1944 in a large Danish clinic, where approximately 2,000 to 3,000 procedures were performed per year. These include antral punctures as well as all other surgical operations. Complications were most frequent following surgical treatment of the ethmoid sinuses. Twenty-four cases of the last-named group are assembled in tabular form showing the time of appearance of the complication, the nature and the order of succession of the symptoms, the course, etc. Disturbance of ocular motility was the most frequent complication, occurring in 19 of the 24 cases. Exophthalmos was seen in 14 cases and lesions of the optic nerve in 14. The amaurosis that developed in most of these cases was permanent. In some of the cases the pathogenesis was established as consisting in direct trauma occurring after perforation of the bone and the periorbita. The author does not believe that anatomic dehiscences are as common as suggested by Onodi and Sluder. He attaches significance to the fact that in 5 of his own cases the lesion was on the right side. A right-handed operator is likely to exert greater pressure with the instrument when working on the right ethmoid bone than on the left. The author quotes MacKenty's proposal that when amaurosis supervenes, the orbit should be opened widely as soon as possible, in order to remove the hematoma which is usually present and causing pressure on the optic nerve.

30. Gallo, A. G., and de Castro, M. D.: Repercussão das affecções dos seios da face e das amígdalas em oftalmologia, Rev. paulista de med. **26**:334 (June) 1945.

31. Godtfredsen, E.: Orbital Complications Following Operations on Paranasal Sinuses, Acta ophth. **22**:401, 1944.

Muzzio [32] makes the following observations in summarizing his experience in 9 cases of orbital complication of sinus infections:

1. The anatomic relations and the structure of the sinuses influence markedly the origin, the development and the spread of orbital complications.

2. The complications differ in extent and gravity according to the point of origin as between the anterior and the posterior ethmoid group.

3. In most cases the common cold or grip is the inciting factor.

4. Polysinusitis is by far more frequent than monosinusitis.

5. Accurate diagnosis requires the help of the oculist, since the status of the fundus is extremely important in the localization of the infection.

6. The oculist should confer with the rhinologist not only on the diagnosis but also on the decision as to operative procedure.

7. In acute cases treatment should be limited to general measures plus simple drainage. Extensive surgical exenteration should be deferred until a safe interval after the acute stage.

Pereira and Tolosa [33] report a case of long-standing suppuration of the conjunctival sac of a man of 40. The lower canaliculus lacrimalis was patent to injected salt solution, but the upper canaliculus was the site of a dacryoethmoid fistula, demonstrated in the roentgenograms after passage of a Bowman probe. Rhinologic examination revealed a chronic infection of the maxillary and ethmoid sinuses, which were cleaned out by a transantral ethmoidectomy of the Ermiro de Lima type.

Torres-Luquin [34] describes the various ocular disturbances which may result when the orbit is invaded by a tumor having its origin in a sinus. The location of the pain, the direction of the bulbar displacement, the involvement of the lacrimal apparatus, the disturbances of vision, etc., may in one way or another point to the sinus giving rise to the tumor and assist in the diagnosis. In every case in which the oculist is confronted with a probable tumor of the orbit it is important that the sinuses be thoroughly investigated.

Kern [35] reports 2 cases in which optic neuritis of children aged 8½ and 10 cleared up under local treatment of infected sinuses. In 1 case hemolytic streptococci and staphylococci were recovered from

32. Muzzio, J. C.: Complicaciones orbitarias de las sinusites: Presentación de 9 observaciones, Rev. san. mil. **45**:186 (Feb.) 1946.

33. Pereira, R. F., and Tolosa, E. E.: Dacriosinusitis simulando una dacriocanaliculitis, Semana méd. **2**:893 (Dec. 6) 1945.

34. Torres-Luquin, P.: Las afecciones neoplasicas de las cavidades paranasales y sus complicaciones orbito-oculares, Rev. mex. de cir., ginec. y cáncer **14**:75 (March) 1946.

35. Kern, E. C.: Sinusitis and Toxic Neuritis, Eye, Ear, Nose & Throat Monthly **25**:34 (Jan.) 1946.

sinus washings. In addition to removal of adenoids and stumps of tonsils, the therapy carried out included irrigation and the local use of sodium sulfathiazole; sulfadiazine was given internally. Both patients were treated for a period of five to six months.

Fishman and Lebo [36] observed orbital cellulitis due to maxillary sinusitis in a man of 27. Exophthalmos, diplopia and slight limitation of ocular movements were present. Later a small tender resistant mass was located on the orbital floor, slightly posterior to the inferior osseous margin of the orbit. Penicillin therapy failed to halt the process, and although irrigation of the antrum yielded a foul greenish pus, the ocular symptoms failed to subside until the antrum was opened and the diseased mucosa and bone were removed.

PULMONARY AND OTHER COMPLICATIONS

Mounier-Kuhn [37] contributes an exhaustive article on the subject of bronchiectasis associated with ethmoantral disease. Clinically, from the rhinologic side he recognizes three types of sinus involvement. The most common type, comprising about two thirds of all cases of sinus involvement, exhibits a bilateral inflammatory state with abundant nasal secretion and characteristic nasal findings. This is severe congestion of the mucosa of the internal surface of the middle turbinate, whereas the external surface is grayish and edematous, as is the rest of the middle meatus. The remainder of the nasal cavity appears more or less normal except possibly for the presence of polypi. The tonsils are usually enlarged, and as a rule there is some cervical adenopathy. The group of patients displaying this type of sinus involvement, as a rule, are those who have received little or no therapy. The second group consists of patients who have been treated or who have had the benefit of a change of climate. Here one will find less secretion, but the appearance of the middle turbinate will still suggest the difference of color just described. The third group is characterized by the presence of large numbers of polypi, more frequently in older persons than in the young children. The thing that is most impressive about all these cases is that the findings persist over long periods despite energetic treatment. The patients present, in addition to the pulmonary changes demonstrated in the roentgenograms, the characteristic syndrome of sinobronchial disease. Also, the author has noted certain changes in the aural apparatus which he believes worthy of comment. In a fairly large proportion of cases these consist of the presence of

36. Fishman, V. P., and Lebo, C. P.: Orbital Cellulitis Secondary to Maxillary Sinusitis: Report of Case, Bull. Pract. Ophth. **16**:41 (Dec.) 1946.

37. Mounier-Kuhn, P.: Le syndrome "ethmoïdoantrite et bronchiectasies" clinique: Etiologie; hypothèses pathogéniques, Ann. d'oto-laryng. **12**:387 (July-Sept.) 1945.

cerumen, bilateral otitis media and general reduction of hearing. In addition, the author mentions trophic changes of the finger nails, occasional disturbances of dentition, absence of the xiphoid process in some cases, and the Kartagener triad of visceral inversion, bronchiectasis and nasal polyposis as indications pointing to a congenital basis of the syndrome.

After discussing the various theories of pathogenesis, he comes to the conclusion that there must be a congenital lack of tissue resistance in these patients, which accounts for the simultaneous infection of both portions of the respiratory tract and the subsequent development of the chronic processes. He cannot concede that the infection is either ascending or descending. One thing which he mentions in support of the theory that there is congenital weakness of tissue is the finding of new bronchial dilatations in previously unaffected areas after lobectomy.

Dutton and Fuchlow [38] point out the differential signs that distinguish the asthma of the sinobronchial syndrome from true atopic asthma. Essentially, the former is characterized by: onset with a cold, gradual development, longer duration, often fever, poor response to epinephrine, neutrophilic nasal smears, marked roentgen findings in chest and sinuses and response to changes of weather. They recommend roentgen therapy for the chronic stage. The doses recommended are 400 roentgens (r), to be applied over the sinuses, and 800 to 1,400 r, to be directed to the chest, in divided doses. Improvement is noted over a period of four to six weeks. A follow-up roentgenologic study is made at the end of six weeks.

Arborelius [39] calls attention to the observation that chronic pulmonary conditions in which cough is the chief symptom are closely associated with chronic sinus disease. In many cases, the nasal symptoms are minimal, and frequently the primary condition is overlooked. Several cases are cited to show the importance of careful examination of the suspected sinuses.

Delp [40] reports a typical case of the Kartagener triad. The patient, a man of 22, had a cough of many years' standing, and roentgenograms revealed chronic bronchiectasis, complete transposition of the intrathoracic structures and chronic pansinusitis with absence of the frontal sinuses. The author states that 46 cases of this kind have been reported in the literature and accepts the theory that congenital maldevelopment is one of the factors in the genesis of bronchiectasis.

---

38. Dutton, L. O., and Fuchlow, J. K.: Sino-Bronchial Syndrome Complicating Atopic Asthma in Children, Ann. Allergy **3**:447 (Nov.-Dec.) 1945.

39. Arborelius, M.: Sinus lungsyndromet, Nord. med. **13**:453 (Feb. 7) 1942.

40. Delp, M. H.: Kartagener's Triad: Situs Inversus; Absent Frontal Sinuses with Maxillary, Ethmoid and Sphenoid Infection, and Bronchiectasis, J. Kansas M. Soc. **47**:93 (March) 1946.

Woodward [41] discusses the problem of the surgical treatment of the sinuses in cases of infectious asthma. He holds that when the diagnosis of chronic sinusitis has been established, it is important to eradicate the focus as completely as possible, even though ill advised or poorly performed operations have aroused resistance in many members of the medical profession. Surgical treatment may not cure asthma in all cases, but when it fails it is still worth while to the extent that it relieves some of the symptoms, such as pain, nasal obstruction and discharge.

Robin [42] discusses the focal infection theory in connection with various rheumatic disorders. He states that the diagnosis of sinusitis must be complete to justify surgical intervention. He therefore lists the findings which are necessary: a history of several undoubted attacks, congestion and secretory activity of the nose, nasopharyngeal streaks of discharge, cloudiness on transillumination, positive proof puncture and definite roentgenologic findings. Conservative treatment of the sinuses may be tried for a while, but if not promptly effective, it should give way to a surgical procedure, which must be complete.

OSTEOMYELITIS

Lillie [43] points out the significant differences between osteomyelitis of the frontal bone and that of the maxillary bone due to differing anatomic features. In the frontal bone infection spreads readily by way of veins of the diploe as well as by communicating veins of both the periosteum and the dura. In the maxillary bone free anastomosis of the terminal branches of the internal maxillary artery insures the integrity of the bone except when the main artery is involved. However, Lillie has observed that in osteomyelitis of the superior maxilla the infection frequently localizes in the region of the lacrimal sac, and investigation has shown that the infection spreads to this area by way of a plexus of veins which surrounds the lacrimal duct, which is subject to trauma in many intranasal antrostomies as well as other types of surgical alteration of this vicinity. Fatalities are due to the infection being carried to the endocranium from the lacrimal veins draining ultimately into the cavernous sinus.

In all cases it is important to identify the bacterial invader, since in some instances an anaerobe may be the offender. In those cases antibiotics would fail. In all cases in which gram-positive organisms

41. Woodward, F. D.: Surgical Management of Infectious Asthma, J. Allergy **17**:260 (Sept.) 1946.

42. Robin, I.: Focal Sepsis: Rhinological Point of View, Rheumatism **3**:25 (July-Sept.) 1946.

43. Lillie, H. I.: Osteomyelitis of the Maxilla Secondary to Suppurative Maxillary Sinusitis, Ann. Otol., Rhin. & Laryng. **55**:495 (Sept.) 1946.

are the invaders, therapy is directed toward adequate administration of penicillin and sulfonamide compounds, and surgical treatment is restricted to simple drainage when pus is located. Extensive resection of bone is not indicated to the same degree in osteomyelitis of the maxilla as in that of the frontal bone. Aside from the evacuation of local accumulations of pus, the only surgical procedure found necessary was the removing of sequestrums later in the course of the disease.

Rojskjaer [44] presents a fairly exhaustive review of osteomyelitis of the frontal bone, discussing anatomy, pathogenesis, bacteriologic findings, symptoms, diagnosis and therapy, with liberal citations of the literature. His 3 cases, which are fully detailed, demonstrate the errors one is prone to commit in misjudging the extent of the process and applying inadequate surgical treatment. In case 1 the patient had previously undergone bilateral radical maxillary sinusotomy as well as bilateral intranasal ethmoidectomy before manifesting the symptoms of osteomyelitis. In this case seven successive operations were performed on the frontal, parietal and squamous bones before the disease was halted. Twice an epidural abscess was uncovered. The patient eventually recovered, although with a very noticeable cranial deformity.

In case 2 osteomyelitis of the frontal bone followed an acute general infection without marked evidence of sinusitis, and the patient had to be operated on three times before the infection was controlled. This patient had an epidural abscess as well as a large abscess of the frontal lobe, which was successfully drained. The author believes this osteomyelitis to have been of hematogenous origin.

In case 3 the osteomyelitis was metastatic from a phlegmon of the arm. Here, despite a rapid course, the outcome was most agreeable, due, no doubt, to the fact that the only operation performed was sufficiently radical to completely eliminate the diseased focus.

De Kleyn [45] discusses the role played by Brechet's veins in the spread of osteomyelitis and the wide variability of involvement. He cites an unusual case: In an infant of 11 months a spreading edema of the eyelids and the scalp developed about a week after the onset of a cold. The edema, which was of the pitting type, extended all the way back to the occiput and was soon followed by extensive subperiosteal suppuration, with necrosis of whole areas of the scalp. The exposed underlying areas of the occipital, temporal and parietal bones were likewise necrotic. Subsequently, sequestrums were removed,

44. Rojskjaer, C. E.: On Osteomyelitis Frontalis Diffusa, with Special Attention to Its Relation to Sinusitis Frontalis, Acta oto-laryng. **34**:445, 1946.

45. de Kleyn, A.: The Connection Between Osteomyelitis of the Cranial Bones and Inflammatory Processes in the Nose and Its Accessory Sinuses, Acta oto-laryng. **34**:534, 1946.

exposing areas of undamaged dura. Eventually all the involved areas regenerated spontaneously, leaving only a small bald spot on the top and back of the head. The author believes that the superior longitudinal sinus had become infected originally by way of communicating veins from the nose.

He also reports a case of osteomyelitis of the frontal bone metastatic from an abscess of the liver of a man of 37. In this case the infection of the frontal sinus was secondary and not primary. The case ended fatally.

Rasmussen [46] reports 2 cases of gangrenous destruction of the facial bones, both of which ended fatally, which in many respects resemble cases previously reported as instances of malignant granuloma. The first case was that of a woman of 24 who gave a history of chronic nasal discharge. Recently she had begun to have pains in the cheek. Both antrums yielded a little pus on puncture, but the condition became rapidly worse, so that the antrum on one side had to be opened. The mucosa was necrotic, and the entire ethmoid bone was soft and mushy. The process extended rapidly, involving both sides of the face. Autopsy revealed multiple abscesses of the lung and destruction of the nasal septum, the turbinate bones, the ethmoid bones and portions of the superior maxilla.

The second case concerned a man of 41. The process began in the hard palate and resembled gumma. However, the history, the serologic tests and provocative antisyphilitic therapy all failed to indicate syphilis. The necrotic disintegration spread through the hard palate into the nose, affecting the septum, the bony nasal bridge, the ethmoid bones and the superior maxilla. Therapeutic measures, including the use of antibiotics and roentgen rays, were futile.

The author cites cases previously reported in the literature and can offer nothing new by way of pathogenesis. The subject is still as mysterious as ever.

Causse [47] reports a fatal case of ostemyelitis of the frontal bone with meningitis and thrombosis of the superior longitudinal sinus. The patient was under observation for two months and was operated on twice. The report is of particular interest because of the illustrations showing a number of sections of the affected bone. These show areas where the blood vessels are thrombosed, areas of resorption and still others where new bone is building. The author demonstrates the pathways of infection via the vessels of the diploe and the dura, as well as the reparative processes going on simultaneously.

---

46. Rasmussen, H.: Osteomyelitis necroticans faciei, Acta oto-laryng. **33**: 254 1945.

47. Causse, R.: Lésions osseuses histologiques dans l'ostéomyélite diffuse des os du crane, Ann. d'oto-laryng. **13**:133 (March-April) 1946.

Morales [48] reports a rare case in which osteomyelitis of the sphenoid bone of a boy of 14 was observed over a period of three years and in which the symptoms and the findings were so indefinite as to delay the final diagnosis for almost two years. The original attack followed "the grip," and the symptoms were peritonsillar abscess, edema of half of the face, the neck and the skull, meningeal symptoms and papillary edema. A tumefaction occurring in the parotid region was incised without obtaining pus, but facial paresis and parotid fistula resulted. Roentgenologic and neurologic examinations failed to localize the lesion. After a remission of several months' duration there was recurrence of the original symptoms with addition of exophthalmos. There were several other remissions at intervals of seven to eight months, apparently due to the administration of sulfonamide drugs and penicillin. It was only after the process had continued for two years that roentgenograms revealed definite clouding of the posterior sinuses on the affected side. A fistula had developed at the external angle of the orbit, and the patient had expelled several sequestrums by blowing his nose.

Penicillin solutions were injected in the temporal zone, subperiosteally and deeply along the external wall of the orbit. Reduction of the exophthalmos followed this treatment, and the patient experienced great relief. The author expressed the opinion that "for the first time in the three years of evolution, I can declare that the patient is in a way of complete recovery."

Munson and Zukerman [49] treated a man of 48, whose symptoms began fifteen days previously. He had pain, purulent nasal discharge and bleeding from the nose. He had had three days of penicillin therapy following the onset, which was continued for a total of five days; he then seemed sufficiently improved to be released. Symptoms recurred within a few days, at which time he was first seen by the authors. Examination showed swelling above the eye, slight elevation of temperature and cloudiness on transillumination. Roentgenograms revealed destruction of the anterior wall of the frontal bone and pansinusitis. A trephine opening released considerable thick pus. Continuous intramuscular drip injection of penicillin solution was started. A week later an external operation was performed, releasing a sequestrum and some diseased mucosa of the frontal sinus. Almost the entire anterior wall was removed. The sinus communicated with its fellow, which was also cleaned out. The injection of penicillin solution was continued for a week. Sulfadiazine was given for several days; then penicillin

48. Morales, E.: Osteomielitis del esfenoides, Rev. otorrinolaring. **6**:1 (March) 1946.

49. Munson, F. T., and Zukerman, M.: A Case of Osteomyelitis of the Frontal Bone Extending from the Frontal Sinus, Complicated by Meningitis: Recovery, Ann. Otol., Rhin. & Laryng. **55**:179 (March) 1946.

therapy was again resumed. Meningeal symptoms appeared about two weeks after the last operation. Sulfadiazine and penicillin were then administered for about two weeks more, when all symptoms had cleared. A few months later examination revealed condensing osteitis at the site of excision of bone.

Gross's [50] case was that of a soldier who had been under treatment for over a month at various army hospitals in consequence of an acute infection of the upper respiratory tract. Evidence of involvement of the bone became apparent within a week of the onset, and despite penicillin therapy and incision and drainage of a frontal abscess, the condition became worse. A bilateral radical frontal sinusotomy was performed at another institution, disclosing osteomyelitis and an epidural abscess. Symptoms of cortical irritation ensued: hyperreflexia, jacksonian attacks, aphasia and increased pressure of spinal fluid. When seen by the author the patient was in a stupor, had definite meningeal symptoms, blurring of the optic disks, hemiplegia and Babinski's sign. A wide incision exposed the frontal and parietal areas on the affected side; diseased bone was removed, the dura incised and a large amount of pus evacuated. After drains were placed and the scalp was loosely sutured back, a counteropening was made by occipital craniotomy and a rubber drain inserted in the subdural space. The author believes that this had much to do with the eventual recovery, since he recalls similar cases of subdural abscess in which autopsy revealed pus in the occipital region. This particular patient recovered, and subsequently cranioplasty was performed with insertion of a tantalum plate.

Cirillo's [51] case emphasizes the importance of adequate drainage, demonstrating that antibiotics are inefficacious unless this surgical principle is closely adhered to. Osteomyelitis of the frontal bone developed in a young woman with a history of long-standing chronic pansinusitis. She was operated on at a time when she was semistuporous. The bone was removed over a wide area, and the frontal and ethmoid sinuses were thoroughly cleaned out. Within twenty-four hours hemiplegia developed, and a second operation was done posterior to the original site, disclosing advancement of the osteomyelitic process and a large subdural abscess. Penicillin failed to produce primary closure, and when the wound was reopened, a further accumulation of bloody pus was drained. From then on, recovery was uninterrupted. The patient was treated with penicillin for the entire period of the illness. The bacteria recovered from the sinus were Staphylococcus albus and nonhemolytic streptococci.

---

50. Gross, S. W.: Osteomyelitis of the Frontal Bone and Subdural Empyema with Recovery, Am. J. Surg. **71**:828 (June) 1946.

51. Cirillo, A. A.: Frontal Osteomyelitis with Subdural Abscess and Hemiplegia After Frontal Sinusitis, J.A.M.A. **131**:105 (May 11) 1946.

Hardy's [52] patient came to operation after an illness of more than a month, during which time symptoms of involvement of a frontal sinus temporarily abated with the use of penicillin. Recurrence of swelling and softening and roentgen evidence of bony involvement led to surgical intervention. At this time a subperiosteal abscess was opened on the right side, both frontal sinuses were opened, softened bone was removed, and a subdural abscess leading into the frontal lobe was drained. Transitory hemiplegia (homolateral) followed the operation. Intensive penicillin therapy brought about subsidence of the drainage, and the patient seemed to improve markedly. However, within ten days acute symptoms developed, indicating that the process had recurred or had extended to the opposite frontoparietal region, and operation revealed extensive disease of the bone and marked external pachymeningitis. The frontal sinus on this side was now completely eradicated, and open drainage plus additional penicillin therapy brought about gradual recovery. The offending organism was Staphylococcus aureus haemolyticus. (This is another example of the bacteriostatic influence of penicillin as well as of its masking effect.)

Bergquist [53] adds another to the growing list of instances of osteomyelitis successfully treated with penicillin. In this instance the infection was due to nonhemolytic streptococci and followed radical maxillary and ethmoid sinusotomy. Chemotherapy alone had no effect, but when it was combined with penicillin therapy the infection was limited to such an extent as to necessitate less radical surgical intervention than is usually required. Such intervention as was indicated consisted in the opening of local accumulations of pus and the removal of sequestrums.

Maxwell [54] describes chronic proliferative osteomyelitis of the skull adjacent to the sinuses as an osteogenetic process arising from the mucoperiosteum. Apparently, the stimulus is provided by a low grade infection and may be hastened by surgical trauma. The author believes that separation of the mucoperiosteum from the bone may be a factor. The formation of new bone may progress to the point where the sinus is partially or even completely obliterated and at times may even reach neoplastic intensity. Often it is differentiated with difficulty from osteoma, hyperostosis, osteofibroma, osteitis fibrosa cystica and Paget's disease except in cases in which the history is consistent and accompanied by serial roentgenograms showing the progress of the ossification. The histologic picture varies with the stage of the process. "Osteoid and

52. Hardy, A. N.: Osteomyelitis of the Frontal Bone with Brain Abscess, Canad. M. A. J. **54**:374 (April) 1946.

53 Bergquist, B.: A Case of Osteomyelitis in the Flat Bones of the Skull, Treated with Penicillin, Acta oto-laryng. **33**:306, 1945.

54. Maxwell, J. H.: Chronic Proliferative Osteomyelitis of the Skull, Ann. Otol., Rhin. & Laryng. **55**:719 (Dec.) 1946.

pyogenic granulation tissue may be present, or normal appearing bone trabeculae with only fibrosis of the marrow spaces may predominate."

Several cases, with accompanying roentgenograms, are described.

Robertson [55] reports 2 cases of chronic osteomyelitis of the frontal bone, which are unusual because ocular proptosis was one of the outstanding symptoms. In the first case, in which the process had developed over a period of three years following a severe cold, there was a marked degree of hyperostosis without suppuration or sequestration. In the second case the osteomyelitis was the result of long-standing suppuration of the frontal sinus. Operation revealed areas of rarefaction and sclerosis, together with marked degeneration of the mucosa of both sinuses. Both of these cases revealed the process of nature's effort to wall off the infection through the development of new bone. The author discusses the pros and cons of radical surgical intervention and advises a conservative attitude pending the outcome of the administration of antibiotics.

MALIGNANT TUMORS

Boysen [56] presents an analysis of 80 cases of cancer of the sinuses. The patients were seen at the University Clinic in Oslo, Norway, during a ten year period. The diagnosis, in many cases difficult, is frequently not made until the later stages of the process. Most of the patients were operated on, but only 14.5 per cent survived beyond two years. Glandular involvement and metastases were infrequent. The procedures followed embraced either the Denker, the Caldwell-Luc or an external resection with surgical diathermy, followed in most cases by roentgen ray or radium therapy.

Bursell,[57] using 5 healthy rabbits, injected 10 mg. of methylcholanthrene into the mucosa of the maxillary sinus and found that in 1 rabbit a tumor of malignant nature developed at the site of injection. Sections of the tumor showed dense lymphocytic and leukocytic infiltration, atypical mitoses, changes in cell shape, metachromasia and vacuolation.

Richardson [58] reports 2 cases of adamantinoma of the maxillary sinus, in one of which an atypical histologic picture was presented. In the first case there was recurrence after a Caldwell-Luc operation, and

55. Robertson, E. G.: Chronic Osteomyelitis of the Frontal Bone, Secondary to Frontal Sinusitis, Roy. Melbourne Hosp. Clin. Rep. **16**:55 (Rec.) 1945.

56. Boysen, F.: Maligne svulster i nesen og dens bihuler, Nord. med. **28**: 2685 (Dec. 21) 1945.

57. Bursell, S.: Tumeurs expérimentales du sinus maxillaire chez le lapin, provoqués par le méthylcholantraène, Acta oto-laryng. **34**:482, 1946.

58. Richardson, G. S.: Adamantinoma of the Maxillary Sinus: Report of Two Cases, Ann Otol., Rhin. & Laryng. **55**:914 (Dec.) 1946.

a second operation was performed through an external incision. Roentgenograms revealed the progressive involvement of the bony walls prior to the second operation. The primary result was good, but the case is too recent to permit one to judge the ultimate outcome. The second case also required extensive resection of the superior maxilla, and here, too, the case was a recent one.

Heatly [59] reports 2 cases of adamantinoma of the maxillary sinus, one concerning a woman of 65 and the other a man of 65. In the first patient recurrence was noted six months and a year after radical extirpation and was treated by electrocoagulation. Further recurrence was considered likely. The second patient was also radically operated on, but too recently to permit any observations as to the likelihood of recurrence.

These tumors are potentially malignant, and recurrence can be avoided only by thorough and careful removal of all affected tissues, including adjacent bony walls. Also, it is helpful to maintain a window in the canine fossa for a long time to permit visual examination of the operative field.

O'Keefe and Clerf,[60] analyzing a series of 47 cases of cancer of the maxillary sinus, all fatal, bring out forcibly the fact that cancer of the maxillary sinus is usually seen too late. More than half of this group of patients had symptoms for at least six months before being hospitalized. In order of frequency, the symptoms most commonly found were swelling of the face (44.6 per cent), swelling of the eye (17 per cent), pain of the face (31.9 per cent), epistaxis (27.6 per cent), pain of the eye (12.7 per cent) and nasal obstruction (12.7 per cent). Reference is made to Ohmgren's classification according to location, which has a definite bearing on the rate of growth and the prognosis. It appears that tumors originating in the lateral part of the superior division grow more slowly than those originating in the medial portion of the superior division or the inferior division. As to early diagnosis, it was found that roentgenograms presented evidence of the true nature of the malady in a larger percentage of cases than did biopsy (61 to 44.6 per cent). Palpable cervical nodes cannot be relied on, since they appear infrequently and usually quite late. Concerning therapy, the authors agree that a combination of electrosurgery and irradiation offers the best chance of cure.

---

59. Heatly, C. A.: Adamantinoma of the Maxillary Sinus with a Report of Two Cases, Ann Otol., Rhin. & Laryng. **55**:322 (June) 1946; Adamantinoma of the Maxillary Sinus, Surgery **20**:636 (Nov.) 1946.

60. O'Keefe, J. J., and Clerf, L. H.: Malignant Tumors of the Maxillary Sinus: An Analysis of Forty-Seven Fatalities, Ann. Otol., Rhin. & Laryng. **55**: 312 (June) 1946.

Mathez [61] cites a number of authors, including del Regato, Coutard, Meier de Kaenel and others, to prove that administering high voltage roentgen rays alone in doses of 7,000 to 7,500 r by the fractional method is the procedure of choice in the treatment of cancer of the maxillary sinus. He fails to present any cases of his own.

Looser [62] reports a case in which a fibroma and a squamous cell carcinoma were found as a joint mass in the hard palate adjoining the alveolus in a woman of about 50. The tumor had been known to be present for approximately fifteen years, during which time it was practically quiescent and gave every indication of being a fibroma. Later it began to grow in the portion involving the alveolus, became ulcerated and, on biopsy, revealed squamous cell carcinoma. Most of the upper jaw was removed, and the histologic examination revealed the two types of tumor fairly well separated by a zone of necrotic tissue. After a lengthy discussion, in which the author reviews the descriptions of "mixed tumors" and "combination tumors," both of which were inapplicable in his case, he comes to the conclusion that in this instance the term *Kollisionstumors* (collision tumors) would be the most appropriate. The fibroma appeared first and probably served as a predisposing factor in the development of the carcinoma. He also feels that carious teeth which were in the vicinity may have been a factor in the role of a long-standing irritant.

The patient had a recurrence, with metastases, and died of a hemorrhage.

Jahn and Paz [63] observed osteoid sarcoma originating in the antrum of a 13 year old girl. It was removed through a sublabial approach, although remnants of the mass remained firmly adherent to the walls of the sinus. Unfortunately, the patient disappeared after the period of convalescence, and the contemplated radiotherapy was not carried out.

Ferrington and Mufson [64] describe a giant cell tumor of the frontal bone, occurring in a woman of 55 several months after a moderately severe trauma. The mass had been previously incised, and a moderate amount of drainage ensued, revealing some streptococci. A fistula persisted. Roentgenograms showed localized absorption of both layers of the frontal bone just above the hair line. At operation a soft, friable, avascular, nonencapsulated mass was exposed and removed piecemeal, exposing intact dura. Histologically, it consisted of large numbers of

61. Mathez, J. A.: Le neoplasme malin du sinus maxillaire se traite uniquement par la radio thérapie profunde, J. de radiol. et d'électrol. **27**:25, 1946.

62. Looser, R.: Zentrales Fibrom und Carcinom des Oberkiefers, Acta otolaryng. **34**:379, 1946.

63. Jahn, F. C., and Paz, O.: Cáncer primitivo del seno maxilar en un adolescente, Arch. venezol. Soc. de oto-rino-laring., oftal., neurol. **6**:66, 1945.

64. Ferrington, E., and Mufson, J. A.: Relationship of the So-Called "Giant-Cell Tumor" to the Frontal Bone, Am. J. Path. **15**:529 (Nov.) 1945.

giant cells, each containing 3 to 12 nuclei, usually clumped in the center, and supported by a stroma of young connective tissue cells. A number of cells containing various amounts of lipoid material were present. In discussing the pathogenesis of the mass, the authors are inclined to place it in the group designated by Jaffe as "giant cell tumor variants" rather than true giant cell tumor. They believe the growth to be of a granulomatous origin, springing from trauma-induced proliferation of the supporting connective tissue of the bone marrow.

Lachman [65] observed a reticulum cell sarcoma of the frontal sinus in a woman of 43, which failed to yield to radiotherapy after surgical removal and resulted fatally. He did not consider a recent trauma or an alleged chronic infection as the probable etiologic factor. The mass was the size of a plum and presented at the internal angle of the orbit in the region of the floor of the sinus, causing some ptosis, and was slightly tender. When exposed it was found to have eroded not only the external table at the supraorbital ridge but most of the ethmoid partitions into the cribriform plate, exposing a considerable area of dura. The mass was soft and vascular. The author presents a complete description of the morphologic aspects of the tumor, with several good photographs of sections.

Daffas [66] reports a case of squamous cell carcinoma involving the ethmoid cells and the maxillary sinus and invading the orbit. It developed slowly and without pain, appearing first as a tumefaction near the internal canthus. A biopsy specimen was removed through a sublabial approach, and shortly after this the mass was removed in its entirety through a Moure transmaxillonasal approach, in which the orbit was exenterated along with the ethmoid labyrinth and the interior of the antrum. The tumor seemed to have had its origin from the posterior wall of the antrum. Galvanocautery was applied after the operation. The patient was free from evidence of recurrence when seen two years later.

Adenocarcinoma of the sinus is rather uncommon. Munson [67] saw a woman of 49 who had had nasal obstruction and discharge for some time and who had undergone polypectomy, with early recurrence of the growth. Examination disclosed a foul purulent discharge, and roentgenograms revealed a very dark antrum and ethmoid labyrinth. A Caldwell-Luc operation was done and suspicious-looking friable masses were encountered in the ethmoid region, extending back to the sphenoid bone. Histologically, it was shown to be an adenocarcinoma, grade III.

65. Lachman, J.: Reticulum Cell Sarcoma, Acta med. orient. **5**:50 (Feb.) 1946.

66. Daffas, J.: Cancer de l'éthmoide avec propagation au sinus maxillaire et a l'orbite, Ann. d'oto-laryng. **13**:45 (Jan.-Feb.) 1946.

67. Munson, F. T.: Adenocarcinoma of the Ethmoid Sinus, Alexander Blain Hosp. Bull. **5**:21 (Feb.) 1946.

A week later an external sphenoethmofrontal exenteration was performed, followed by surgical diathermy. There was no recurrence up to the time of publication (fourteen months).

Collenza [68] reports a case of adenocarcinoma of the sphenoid sinus. The patient was a woman of 50. The growth had invaded the orbit, causing marked exophthalmos and fixation of the bulb with loss of vision. Also the tumor extended forward into the ethmoid cells and the frontal sinus and eroded superiorly into the frontal lobe of the brain. It was so extensive when first seen as to be inoperable and was unsuccessfully treated with radiation.

Goldman [69] reports the case of a man of 53 in whom, within a period of a few weeks, the following symptoms developed: headache and ophthalmoplegia without impairment of visual acuity. Roentgenograms revealed no abnormality at first and could not be obtained at a later date because of the confused condition of the patient, who rapidly became comatose and died. Autopsy revealed a grayish red mass, 2.5 by 0.8 cm. in size, lateral to the sella turcica and displacing the cavernous sinus. Sections of the sphenoid bone showed the entire bone replaced by tumor tissue, which extended into and filled the sinus. The cavernous sinus was compressed and the contents of the carotid canal were displaced. The nerves were unusually cellular, there being an increase both of Schwann cells and fibroblasts. The tumor sections showed irregular bundles of flat cells with oval nuclei, and there were quite a few mononucleated and multinucleated giant cells. The photographs of the sections were unusually clear. Discussing the clinical course, the author stresses (1) the syndrome and (2) the unexpected finding. The simultaneous or successive involvement of the third, fourth, sixth and first division of the fifth cranial nerve indicated a lesion in the lateral wall of the cavernous sinus or the sphenoid fissure. Proptosis and orbital edema are supposed to be associated with lesions of the cavernous sinus, yet in this case they were absent, even though the sinus was completely obliterated by pressure. Fibrosarcoma of the cranial bones is rare, only 2 cases having been found by the author; in one the parietal and in the other the occipital bone was involved.

Hurteau [70] reports 2 cases of meningioma invading the sinuses. One was a typical meningothelial meningioma with whorl formation. The other was of a fibroblastic type. He believes that this tissue originated from meningothelial cells which "have the capacity to differentiate into

68. Collenza, D.: Adeno-carcinoma del seno sfenoidale, Atti d. r. Accad. d. fisiocrit. in Siena (1944) **12**:32, 1944.

69. Goldman, M.: Fibrosarcoma of the Sphenoid Bone, Producing the Syndrome of the Lateral Wall of the Cavernous Sinus: A Case Report, J. Neuropath. & Exper. Neurol. **5**:155 (April) 1946.

70. Hurteau, W. W.: Invasion of Paranasal Sinuses by Tumors of Meningeal Origin, Am. J. Orthodontics **32**:556 (Sept.) 1946.

cells that form fibroglia fibrils, lay down collagen and ultimately become morphologically indistinguishable from fibroblasts."

In case 1 the growth was removed from the olfactory groove through a hypophysial approach; it recurred after three years and invaded all of the sinuses, causing severe recurring hemorrhages. Operation was refused for several years, until the patient was lost sight of.

In the second case, that of a man of 53, the meningioma involved the left side of the face, causing marked asymmetry, and in the roentgen films was shown to involve the frontal, ethmoid and sphenoid sinuses, extending into the base of the skull and eroding the dorsum sellae turcicae and posterior clinoid process. The eye was enucleated, exposing a mass that protruded from the ethmoid labyrinth. The patient died six days later. Autopsy revealed the extensive nature of the growth.

The histologic sections shown in the article are clear and exceedingly instructive.

NONMALIGNANT TUMORS

Nascimento [71] reports a case of osteoma of the frontoethmoid region, complicated by an intracerebral pneumatocele. The patient was a man of 24. The pneumatocele extended backward to the parietal lobe, causing sufficient pressure to result in contralateral paresis of the face and of the arm. The patient also suffered from severe headaches and projectile vomiting. There was no record as to whether or not the man was operated on.

Malgeri [72] reports a case of chondroma involving the ethmosphenoid region in a man of 48. It had developed slowly over a period of years, eventually attaining sufficient size to obstruct one side of the nose completely. Posteriorly the growth invaded the floor of the sella turcica, and externally it eroded into the orbit, causing proptosis with gradual loss of vision from pressure on the nerve. Several illustrations showing the histologic structure of the growth are presented.

Piergrossi [73] describes the clinical and radiologic aspects of osteomas of the skull, which have a predilection for the region of the orbit and the sinuses. Hyperostoses are at times not to be distinguished from osteomas because they present a similar roentgenographic picture. Perisinusitis hyperostosis is, according to the author, an early stage of leontiasis ossea, which he believes is due to an infection of the lymphatic

71. Nascimento, H.: Osteoma fronto-etmoidal e pneumatocele, Arq. Inst. Pen. Burn. **7**:161 (Dec.) 1945.

72. Malgeri, G.: Contributo allo studio degli encondromi etmoido-sfenoidali: Significato nosologico e diagnosi radiologica, Ann. laring., otol. **42**:41, 1942.

73. Piergrossi, A.: Osteomi ed iperostosi nel territorio dei seni paranasali, Quaderni radiol. **7**:139, 1942.

channels of the bone and may have its origin in the sinuses. It is easily distinguished from Paget's disease by the age, by the location and, mainly, by the absence of a convex base (*connessibasia*), also by the presence of the typical facies.

Duclos and Corcuera[74] report an osteoma involving the ethmomaxillary region in a girl of 18, whose symptoms came on shortly after a thyroidectomy. The exophthalmos which was present prior to this operation persisted and was followed by some displacement of the bulb. Roentgenograms revealed a dense shadow at the inner angle of the orbit. The mass was removed through a curved incision and was found to involve the ascending process of the maxilla and to occupy most of the ethmoid labyrinth. The lamina papyracea was completely destroyed, and the tumor projected into the medial portion of the orbit. The authors believe that the tumor had its origin in the vicinity of the maxillolacrimal suture.

Linthicum, Rand and Reeves,[75] in a well written article, present in detail a case of mucocele of the sphenoid sinus. The patient was a man of 54. There was a long history of previous sinus trouble, with operations on the frontal and maxillary sinuses in 1923, 1930 and 1931. Despite these procedures, the patient had severe attacks of left frontal and occipital headache, general apathy and one or two spells of unconsciousness. Roentgenograms of the skull revealed erosion of the sella turcica with disappearance of the clinoid processes. A diagnosis of suprasellar cyst or meningioma was made, and a frontotemporal osteoplastic craniotomy was performed. This revealed an irregular bluish mass about 4 cm. in diameter, which was aspirated, yielding about 9 cc. of thick, greenish brown material which contained pus and red cells, a few leukocytes and some cholesterol crystals. The cystic mass was opened and the content aspirated. It seemed to extend down into the sphenoid sinus. The incision was closed and a piece of muscle covered the small defect. The vision, which had been deteriorating, remained bad, and examination of the fundus showed old perimacular chorioretinitis. The sphenoid sinus was then opened intranasally and it was found that the entire area was membranous, there being scarcely any bone to be felt in any direction. The cavity was aspirated several times and continued to drain spontaneously. The patient's condition did not improve. Headaches were followed by jacksonian seizures, and the spinal fluid revealed increased pressure, 105 white cells per cubic millimeter and 135 mg. of protein per hundred cubic centimeters. The

74. Duclos, J., and Corcuera, J. M.: Osteoma etmoidomaxillar, Rev. otorrinolaring. **5**:152 (Sept.) 1945.

75. Linthicum, F. H.; Rand, C. W., and Reeves, D. L.: Mucocele of the Sphenoid Sinus: Report of a Case with Autopsy Findings, J. Neurosurg. **3**:444 (Sept.) 1946.

meningeal symptoms increased, with signs of increasing intracranial pressure, and the patient succumbed about eighteen months after the first cranial operation. Autopsy revealed the mucocele occupying both sphenoid sinuses and measuring 5 by 2.5 by 2 cm. Also, an abscess of the frontal lobe was found, measuring 4.5 by 3 by 5 cm., with a fairly well defined wall.

A discussion of the cause and the pathogenesis, with citations from many sources, adds much to the interest of this unusual case, as do the excellent illustrations.

Goodyear [76] recommends opening mucoceles through the nose, removing the lower portion of the projecting mass, splitting the middle turbinate if necessary, and leaving the lining of the sac intact. He claims that this smooth lining, which is epithelium, is as good as any that might grow in, and insures a clean, nonsecreting cavity. If the mucocele has bulged into the orbit, it can be reduced by finger pressure once it has been drained into the nose.

He also agrees that in severe acute infections of the frontal sinus it is safe as well as practical to trephine through the floor of the sinus for drainage. Continuing further on the subject of lesions of interest to the ophthalmologist as well as the rhinologist, he calls attention to subperiosteal abscess occurring at the inner angle of the orbit, due usually to infection of the ethmoid labyrinth, which should be incised simply for drainage, without any attempt at wide curettage of the cells. Also, he mentions Sewell's operation for malignant exophthalmos, in which the lamina papyracea is removed as far back as the optic foramen. Corneal ulcers in his experience are often due to chronic infection of the antrum. Roentgen studies made with iodized poppyseed oil 40 per cent will show a thickened lining of the sinus. A properly executed intranasal antrostomy should shorten the course of the disease.

Further, the author calls attention to the fact that patients with optic neuritis frequently recover promptly after surgical opening of the ethmoid and sphenoid sinuses. He has seen the same spectacular results follow instillation of iodized oil.

An unusual bilocular cyst of the antrum is reported by Sargnon.[77] One lobe filled the antrum, while the other occupied the soft palate, the two sacs being connected by a tube which passed through an opening in the floor of the sinus about a centimeter or more in diameter. It was removed via a sublabial incision and was found to contain

76. Goodyear, H. M.: Modern Treatment of Mucocele of the Frontal and Ethmoidal Sinuses: Some Comments on the Treatment of Lesions Pertaining to the Orbit and Adjacent Nasal Sinuses, South. M. J. **39**:42 (Jan.) 1946.

77. Sargnon, A.: Volumineux kyste sinuso-maxillaire et palatin en bissac avec perforation osseuse palatine: Cure radicale sinuso-maxillaire; guérison complète, Oto-rhino-laryng. internat. **29**:161 (Nov.) 1941.

a nonpurulent brownish fluid. Histologically, some dense fibrotendinous fragments were found, with some blood pigment; also osseous lamellas and osteoclasts. The mucous glands were hypertrophied, and there was considerable inflammatory exudate. There was no evidence of any dental origin. The author believes the etiologic factor to have been traumatic, since the patient gave a history of having been in an automobile accident some years before, at which time the stem of a pipe he was smoking was forcibly projected against his palate.

Bey [78] describes a cyst formed in the floor of the nose, often mistaken for a dentigerous cyst. It is known variously as a "mucous cyst, cyst of the alae nasi, cyst of the entrance of the nose, vestibular cyst," etc. The author favors the last-named term. He states that he has observed 10 cases of this type, one of which is now reported. Several roentgenograms show the cyst, filled with injected iodized oil, resting in the lateral wall of the ala nasi, well defined and easily distinguished from a possible dentigerous cyst.

Myers [79] reports 3 cases of choanal polyp of antral origin discovered in a three year period, during which over 25,000 otorhinologic examinations were made. In all 3 instances the mass was removed by a Caldwell-Luc operation. In case 1 the tumor filled the sinus and extended via the ostium to the nasopharynx. The mucosa was degenerated. In case 2 a similar condition was encountered, and the nasopharyngeal extension of the mass was removed by means of a tenaculum via the oral route. In case 3 several polypoid masses were found in the sinus, one of which extended through the ostium to the nasopharynx.

Passe [80] describes a case in which osteitis fibrosa occurring in a man of 20 was treated by total excision of the superior maxilla via the sublabial approach. After preliminary ligation of the external carotid artery, it was found possible to expose the face of the maxilla by dissection of the soft tissues upward from a sweeping sublabial incision. The frontal process was cut through with bone forceps and drill. The malar bone was divided by a gigli saw introduced through a small external opening, and the hard palate, by means of a sharp cutting burr. The final attachments to the pterygoid process and lateral mass of the ethmoid bone were easily broken off by lion forceps. There was little bleeding. On the twelfth day the raw surface was covered by an intermediate graft taken from the thigh and maintained in place by an obturator previously constructed, which was held firmly in place by

78. Bey, A. S. H.: Vestibular Cysts, J. Laryng. & Otol. **61**:215 (March) 1946.

79. Myers, D.: Obstruction of the Nasopharynx Secondary to Choanal Polyp of Antral Origin, Arch. Otolaryng. **44**:328 (Sept.) 1946.

80. Passe, E. R. G.: Excision of Superior Maxilla and Ethmoid Labyrinth, Arch. Otolaryng. **43**:379 (April) 1946.

a rod attached to a plaster head cast. The final prosthesis is shown, adequately filling the defect, with a good cosmetic result.

Robert and Forman [81] state that odontoma is rare, less than 50 cases having been reported in the literature. Their case concerned a boy of 12 with a history of swelling of the cheek of three months' duration. Roentgenograms revealed two displaced teeth, one near the orbital floor and another near the inferior meatus. A third tooth was found at operation. These teeth were contained in a cystic cavity, which had eroded the anterior wall of the sinus. The entire mass was removed via a Caldwell-Luc operation.

(*To Be Continued*)

81. Robert, S. E., and Forman, F. S.: Follicular Odontoma of the Maxillary Sinus, Ann. Otol., Rhin. & Laryng. **55**:175 (March) 1946.

Abstracts from Current Literature

Ear

HEARING AID CLINICS IN ENGLAND. A. W. G. EWING, Laryngoscope **57**:41 (Jan.) 1947.

Ewing describes briefly the hearing aid clinics of England. One hopes that sometime he will present a more complete account, as many questions are left unanswered.

The hearing aid clinics are government connected. There are three large ones and numerous small ones, affiliated with various hospitals. In two of the three large ones small fees are collected; the third is free. Although veterans are not neglected, the main purpose of the clinics is to care for deafened civilians. Pure tone and speech reception tests are used. Ewing does not state whether the latter are monitored and does not go into detail as to the methods of examination and testing. Hearing aid instruction and lip-reading classes are found at the clinics. Interesting figures are presented as to the hearing gains in 100 cases with an average loss of 62 decibels. In this group there was 21 per cent accuracy in hearing certain test sentences with no aid whatsoever. With lip-reading training alone, and with a hearing aid but no lip-reading training, the accuracy was increased to 64 per cent. With both lip reading and a hearing aid the accuracy increased to 90 per cent.

HITSCHLER, Philadelphia.

SYMPOSIUM ON NOISE: NOISE IN INDUSTRY. W. E. GROVE, Laryngoscope **57**:114 (Feb.) 1947.

Excessive noise has long been recognized as a cause of loss of hearing. World War II, together with its industrial expansion, increased the importance of this problem.

Noise, particularly that of high pitch, has most effect on the basal coil of the cochlea, producing a high tone loss. The reasons for this are discussed.

There are many factors that must be recognized when one tries to evaluate the effect of factory noise on the individual ear. Only when an accurate examination of hearing was made before the subject embarked on his noisy occupation can a satisfactory estimate be made of the effects of the occupation itself.

Means of reducing the noise of an occupation are suggested. They range from changes in the design of the noisy machinery to various devices for isolating and absorbing sound. When other means fail, ear defenders should be worn.

HITSCHLER, Philadelphia.

SYMPOSIUM ON NOISE: EAR DEFENDERS. H. MARSHALL TAYLOR, Laryngoscope **57**:137 (Feb.) 1947.

In recent years the importance of insulating the hearing organ against loud noises has been recognized. A defender, known as the "V-51R warden" has been designed that is comfortable, provides high acoustic insulation (25 to 40 decibels) and allows speech to be heard through it. Such qualities are essential to a good defender. This defender is recommended as being useful not only in battle but in industry.

HITSCHLER, Philadelphia.

PETROSITIS FROM A PATHOLOGIC AND A CLINICAL STANDPOINT. HÅKAN R. GADOLIN, Acta oto-laryng. **35**:1 (Jan.-April) 1947.

Gadolin describes a case of acute infection of the middle ear in which it was necessary to operate six days after the onset of the condition because of severe zygomatic mastoiditis. On the fifteenth postoperative day petrositis developed with labyrinthitis and meningitis. Gadolin goes into the difficulties of making a diagnosis of such an early stage of petrositis, particularly because of the involvement of the zygomatic cells, and he emphasizes the constant study of the cerebrospinal fluid with special reference to the indications for surgical intervention.

The pneumatization of the mastoid process was restricted, but the zygomatic area and the neighboring squama showed considerable, though irregular, pneumatization. In the pyramid a superior tract of small cells was discovered which had failed to show up in a roentgenographic study.

Gradenigo's symptom complex was present, but the symptoms of the zygomatic mastoiditis so overshadowed those of the petrositis that the diagnosis of petrositis was made only after the occurrence of paralysis of the abducens nerve. In addition to homolateral trigeminal neuralgia and abducens paralysis there were loss of labyrinthine function, paralysis of the facial nerve, contralateral irritation of the trigeminal nerve, contralateral paralysis of the abducens nerve, bilateral loss of corneal reflexes and homolateral mydriasis.

The tract of infected cells was opened from the attic and followed to the apex of the pyramid, but no abscess was encountered at the apex. Because of these operative findings and because of the negative roentgenologic findings, Gadolin finds it difficult to decide whether the affected cranial nerves were damaged extradurally or intradurally. He also finds it difficult to decide whether the labyrinth was involved through the bony capsule or by way of the internal auditory meatus. He is certain, however, that the labyrinthitis and the facial paralysis cannot be charged to the operative procedure. With the exception of the homolateral paralysis of the sixth nerve, which was still present when the patient was dismissed from the clinic, all the remaining symptoms referable to involvement of cranial nerves disappeared, some rapidly, some slowly. The facial paralysis was treated with a plastic procedure utilizing the temporal muscle.

The cure of the patient, with the exception of the operative procedure and the repeated lumbar punctures, was due to intensive administration of sulfapyridine, and Gadolin recommends that intensive sulfapyridine medication be instituted in every case of petrositis in which the spinal fluid pressure is increased or in which the increasing cell count indicates danger of meningeal involvement.

GROVE, Milwaukee.

Pharynx

TONSILLECTOMY IN EARLY CONVALESCENCE FROM ACUTE TONSILLITIS WITH THE PROPHYLACTIC USE OF SULFONAMIDES. JOHN E. BORDLEY and THOMAS McP. BROWN, Ann. Otol., Rhin. & Laryng. **55**:751 (Dec.) 1946.

The authors, finding that many men did not have their tonsils removed, owing to frequent change of station and to the delay usually insisted on before operation, following acute tonsillar infection, decided to undertake operation shortly after the acute episode. They felt that chemotherapy administered for twenty-four hours prior to surgical treatment and for several days after it would prevent serious complications. Many of the men had had repeated acute infections within the first year after they were first seen with the acute involvement, but owing to the usual

delay before surgical treatment, they were transferred, and the treatment repeatedly postponed.

The patients selected for the study were those convalescent from acute follicular tonsillitis, acute infection with Vincent's organisms complicated by beta hemolytic streptococcus infection, and acute sore throat without exudate but with positive beta hemolytic streptococcus cultures. In the study tonsillectomy was performed immediately after the acute infection in 100 cases, with sulfonamide compounds (sulfathiazole and sulfadiazine seemed to work equally well) administered for twenty-four hours prior to surgical treatment and for five days following it. These cases were compared with 100 elective cases in which tonsillectomy was performed but in which no drug was administered. The postoperative course was in general better in the cases in which sulfonamide drugs were given than in the elective cases. One hundred additional patients, most of them convalescent from acute infection, were operated on, with the routine use of sulfonamide therapy, and the postoperative course of these was uneventful. Where there was drug sensitivity, penicillin was substituted with equally good results.

M. V. MILLER, Philadelphia.

CHANGES IN THE COLOR OF THE PHARYNX FOLLOWING SMOKING. A. H. ANDREWS, JR.; C. W. LENTH; E. E. STAUNTON and P. H. HOLINGER, Laryngoscope **57**:211 (March) 1947.

Elaborate studies were conducted to determine the effect of smoking on the pharynx. Cigarets treated with diethylene glycol and with glycerin were compared and found to be similar. The color of the pharyngeal mucosa was probably blanched by the smoke.

HITSCHLER, Philadelphia.

Larynx

SURGICAL TREATMENT FOR CARCINOMA OF THE LARYNX. SIMON JESBERG, J. A. M. A. **134**:121 (May 10) 1947.

The treatment for laryngeal cancer is surgical or radiologic, or it is a combination of the two. The selection must be under the direction of one who is a laryngologist and is radiologically minded or a radiologist who is trained in laryngology.

There is only one Coutard in this world who is a radiologist and a laryngologist combined.

The cancerous growth follows the pattern of the parent cell. In the vocal cord it is almost invariably squamous cell carcinoma of low grade, which is not radiosensitive and belongs to the domain of surgery, though elsewhere in the larynx the exact opposite holds. High grade cancer is more amenable to irradiation than to surgical excision. Cancers of the vocal cord are slow growing, and the bed from which they spring is poorly supplied with lymphatic channels and blood vessels, so that they can be expected to be focalized for a long time. Cancers arising elsewhere in the larynx spring from a bed that is abundantly supplied with lymphatic channels and blood vessels; their growth is rapid, and they extend early to the regional nodes or metastasize.

No cancer can be diagnosed without a biopsy. The Jackson biopsy forceps is used in securing tissue for examination.

It is best to classify the cancer according to its site of origin, whether it is cordal or extracordal. Cordal cancers belong to the domain of surgery. Extracordal cancers arising from columnar epithelium are almost invariably high grade and more radiosensitive.

Coutard tells us: "The more the embryonal or undifferentiated cells, the greater the possibility of curing by irradiation. Cancers consisting of differentiated cells developing on fibroelastic connective tissue are forms generally incurable or difficult to cure by irradiation, because of the lack of radiosensitivity. When they are technically operable, they belong to the domain of surgery, because the cells multiply slowly and have only a slight tendency to disseminate and develop adenopathy rarely or late."

The author draws the following conclusions:

1. Surgical treatment for carcinoma of the larynx consists in laryngofissure with cordectomy if the cord is motile and laryngofissure with removal of underlying laryngeal cartilage if the cord is fixed.

2. Cordal cancer approaching the anterior commissure should be treated by removal of the anterior part of the opposite cord with laryngofissure.

3. Cordal cancer that has involved the larynx beyond the extent of the true cord calls for laryngectomy.

4. If the cancer is extensive or high grade, a preliminary course of irradiation should be given before surgical excision is attempted. GORDON, Philadelphia.

Nose

INTRANASAL MEDICATION. TERENCE CAWTHORNE, Ann. Otol., Rhin. & Laryng. **55**:518 (Sept.) 1946.

Cawthorne studied the relative effect of intranasal medication as achieved by the use of drops, sprays and sniffs and by head-low position. He determined the efficiency by the degree to which the olfactory and other senses were affected when cocaine solution was applied by these methods. His investigation demonstrated that the head-low position was definitely the most effective, and he attributes this to the fact that in this position the fluid used gravitated into contact with the whole of the accessible mucous membrane and was kept in contact with the membrane sufficiently long to have an effective action.

M. V. MILLER, Philadelphia.

IRRADIATION OF HYPERPLASTIC LYMPHOID TISSUE IN THE NASOPHARYNX. SAMUEL J. CROWE and EDWARD M. WALZL, J. A. M. A. **134**:124 (May 10) 1947.

Hypertrophy or chronic infection of lymphoid tissue of the nasopharynx has long been recognized as the cause of many ills related to the upper air passages, such as repeated colds, sinusitis, aural infections and deafness. It is usually impossible to remove nodules of lymphoid tissue completely by surgical operation, and nodules which are left often hypertrophy rapidly.

Fortunately, lymphoid tissue is extremely sensitive to radium or roentgen irradiation.

Two types of applicators are used in the clinic of the authors; one makes use of radon gas and the other of a radium salt, the former having certain advantages.

Normally functioning eustachian tubes are a prerequisite to good hearing. Nodules of lymphoid tissue in or about the eustachian orifices, which become infected with each cold, will cause mechanical blocking and overproduction of mucus in the glands at the mouths of the tubes.

Every child who has been treated should have an examination of his tympanic membranes, nasopharynx and hearing twice a year for two or three years.

There have been some patients who suffered with aerotitis. The latter condition is apt to become more prevalent because of the increase of air travel. Such patients seem to have an inability to equalize the air pressure of the middle ear. It is easy to equalize air pressure in the ascent, but more difficult to do so in the descent. A good many of the patients examined were shown to have adenoidal obstruction of the eustachian tubes. An extensive program of radium treatment was undertaken, and there was uniformly great improvement in a large percentage of the fliers so treated.

This treatment has also been found of value in children and some adults who have had the tonsils and adenoidal masses removed but still have rhinitis and pharyngitis, especially in the allergic ones.

GORDON, Philadelphia.

Miscellaneous

EFFECT OF PENICILLIN LOZENGES ON CLINICAL AND BACTERIOLOGICAL FINDINGS OF PATIENTS. DEKELE TAYLOR, WAVE ELAINE CULVER and W. J. NUNGESTER, Ann. Otol., Rhin. & Laryng. **55**:743 (Dec.) 1946.

Two groups were investigated. In one group, 26 patients, specimens were examined bacteriologically during and after a period of treatment with penicillin lozenges. In this group 11 had undergone tonsillectomy and 5 tooth extraction, 4 were normal controls, and 6 had miscellaneous conditions of the mouth and throat. In the second group, composed of a larger number of patients with acute pharyngitis and acute tonsillitis, clinical observations alone were made in addition to the bacteriologic studies.

Each lozenge contained 500 Oxford units of penicillin in a lactose base. In some cases a similar lozenge containing 1,000 units was used, but in these cases specimens were not examined bacteriologically.

Patients given penicillin lozenges after tonsillectomy appeared to have less membrane formation in the fossae, and they complained less of pain than those not so treated. Similar findings were made in the patients treated after tooth extraction. The lozenges seemed to be of little value in cases of deep-seated tonsillitis or pharyngitis, but seemed to have definite value in cases of pharyngitis at an early stage. The bacteriologic findings supported the clinical observations. The total, and more particularly the Streptococcus viridans, counts of mouth and throat flora were lowered by the use of penicillin lozenges. The pencillin was present in the mouth washings for only a short time after the disappearance of the lozenges.

M. V. MILLER, Philadelphia.

INCIDENCE OF MALIGNANT NEOPLASTIC DISEASE OF THE HEAD AND NECK. JOSEPH G. SCHOOLMAN, Ann. Otol., Rhin. & Laryng. **55**:789 (Dec.) 1946.

The conclusions reached from this statistical survey were:

1. Malignant neoplastic disease occurs most frequently in the sixth decade, with the seventh next in order.

2. Malignant tumors occur seven times more frequently in males than in females.

3. The age span for cancer incidence is approximately the same for the two sexes, but for the male it begins about nine years later than for the female.

4. Most tumors are of the squamous type.

5. Carcinoma of the larynx is closely followed in incidence by carcinoma of the esophagus, and the two make up more than one third of the tumors of the head and neck.

6. The presence of a tumor clinic in an institution will tend to increase the case incidence of malignant neoplastic disease.

M. V. Miller, Philadelphia.

Comments on Testing Aphasics in the Speech Clinic. Clarence T. Simon, J. Speech Disorders **11**:139, 1946.

Information concerning the locus of the lesion is of no value to the speech correctionist. There is no basis in clinical data for the conventional classification of types of aphasia. Any attempt to put patients into these categories or to guide testing and remedial programs on the basis of them militates against substantial results. The specific reason for testing is to discover the specific abilities and disabilities of the patient's existing language process. The best therapy uses the patient's abilities as a basis for training in those skills he needs most for his immediate social and economic demands.

Palmer, Wichita, Kan.

Cosmetic Reduction of Full, Everted Lower Lip. C. Firestone, Northwest Med. **45**:499 (July) 1946.

The author reviews the anatomy of the musculature of the lower lip and describes the technic of his operation. The skin of the lip is undermined through an incision made transversely ½ inch (12 mm.) below the oral angle. An incision is then made transversely through the mucosal surface, ¼ inch (6 mm.) from the fundus of the gingivolabial fossa; the mucous membrane is undermined up to a line which represents the meeting of the mucosal surfaces of the closed lips. The underlying loose areolar tissue, including the small mucous glands, is removed, and the mucous membrane is put on the stretch, anchor sutures being used to secure the desired degree of reduction. Excess mucous membrane is excised, and the wound is closed with interrupted silk sutures, which are removed on the sixth day.

Seltzer, Philadelphia.

Disfigurement of the Face. W. L. Gatewood, Virginia M. Morthly **73**:35 (Aug) 1946.

Gatewood reviews routine operations for: (1) facial paralysis; (2) malformed, protruding ears; (3) relaxation of muscles of the face and the neck with redundancy of the overlying skin; (4) rhinophyma; (5) contracted scars of eyelid, nose and cheek; (6) nasal distortions. No original technic is presented.

Seltzer, Philadelphia

Treatment of Influenzal Meningitis. Conrad Gasser, Helvet. pædiat. acta **1**:165 (Nov.) 1945.

Treatment of influenzal meningitis is discussed and 7 cases in which there was recovery are reviewed. Decisive for these cures was a high dose of the sulfonamide compounds at the beginning of the treatment (0.5 Gm. per kilogram of body weight a day). This dose has to be started as soon as the presence of influenzal meningitis is suspected. Oral administration may be allowed in cases of light disease, but in cases of severe disease parenteral administration is necessary,

eventually combined with continuous intravenous infusion. In case of doubt purulent leptomeningitis must be treated as influenzal meningitis. If the treatment proves to be unsuccessful or if a relapse occurs, the sulfonamide drug must be replaced by another one, again given in the same high dose. The higher the amount of sulfonamide drug per day, the better is the prognosis. In addition, daily spinal taps, repeated blood transfusions, administration of vitamins and stimulation are recommended. The treatment has to be surveyed by regular blood controls. Oncoming toxic manifestations must be counteracted by repeated blood transfusions; furthermore, the drug must be changed. Treatment with sulfonamide drugs may not be discontinued until the examination of the spinal fluid shows a normal result and the temperature has become normal.

AUTHOR'S SUMMARY. [AM. J. DIS. CHILD.]

Book Reviews

Office Treatment of the Eye. By Elias Selinger, M.D. Price $7.75. Chicago: The Year Book Publishers, 1947.

This book is offered as a manual of ophthalmology and was inspired by the needs of resident physicians and postgraduate students. In the words of the author it describes "ordinary office procedures and routine ophthalmological treatments in detail." Chapters are devoted to the diagnosis and treatment of injuries and diseases of the internal and the external eye, carrying the treatment up to minor surgical procedures and stopping short at the major. There is also a chapter on problems of refraction, and another on the extraocular muscles.

Of particular interest is a separate chaper on chemotherapy which presents in well organized form the local and systemic ophthalmologic uses of the sulfonamide compounds and penicillin. Specific applications of these drugs also appear throughout the text. In addition there is an appendix on iontophoresis, a method of treatment infrequently used and not recommended by many ophthalmologists. This presentation, however, suggests that a new evaluation should be made.

As a manual to be placed on the shelf over the nurses' desk at the end of the ward this book will have a function. The ophthalmologist will prefer books giving separate and more exhaustive treatments of the subjects covered.

The most valuable aspect of the book is the reflection found therein of one man's experience and consequent opinions. This is always stimulating whether one's own experience is in agreement or not. For instance, recent experiments on laboratory animals would indicate that sulfonamide drugs retard regeneration of corneal epithelium and increase scarring of the cornea. Dr. Selinger has not found this to be true clinically, and that appears to be the general experience. On the other hand, there is here recommended a much greater use of ophthalmic ointments than meets with universal acceptance. These, many ophthalmologists find, create a film before the eyes, retain secretions, and cause a greasy appearance that ambulatory patients find objectionable.

Headache. By Louis G. Moench, M.D., assistant clinical professor of medicine, University of Utah School of Medicine; internist, Salt Lake Clinic, Salt Lake City, Utah. Price $3.50. Pp. 207, with 58 illustrations. Chicago: The Year Book Publishers, Inc., 1947.

Dr. Moench, an internist, presents a book on the important subject of "Headache." It consists of a summary of researches bearing on this subject, notably those of Wolff and co-workers, Riley, von Storch, Lennox, Horton and others. All of these were incorporated along with the experiences of the author.

There are 10 chapters as follows: I. Pathologic Anatomy and Physiology of Headache. II. Headaches from Intracranial Pathology, Spinal Puncture and Ventriculography. III. Cranial Nerve Neuralgias. IV. Headache of Ocular Origin. V. Headache of Nasal Origin. VI. Headache from Pathology in the Neck. VII. Headache from Systemic Disorders. VIII. Histamine Headaches. IX. Migraine. X. Headache of Emotional Origin. Finally there is an index.

The book consists of 207 pages, with 58 figures and 8 tables. The entire text is clearly written and is informative. The figures are drawings and schemata

which supplement the text matter. The tables serve to simplify the differential diagnosis in given cases. After each chapter a bibliography appears, which enables any reader to study a particular problem further.

The symptom of headache frequently confronts the internist and some of the specialists. It is always of interest to the rhinologist, for this symptom is often encountered in his practice. Sometimes the sinuses are responsible; at times they may relate to it partly, and in many instances they bear no relation at all.

Particularly informative to this reviewer is the clearcut description of the pain-sensitive structures of the head (the author describing in moderate detail the sensitive and the insensitive structures, with emphasis on the cranial vascular structures as the greatest source of pain and an account of a disturbance of the encephalic hydrodynamic mechanism that is responsible for the majority of headaches).

There are several diagrams which might aid the reader in the problematic attempt to separate the neuralgias of the cranial nerves from ocular, otic, rhinologic or dental pain.

Referring to the question of headache of nasal origin, the author states "that headache of true nasal origin comprises only a small percentage of all headaches" and that "sinusitis is not a common cause of headache and the false diagnosis of sinusitis because of headache often leads to much unnecessary and often meddlesome treatment." Farther on the author states: "Most periodic headaches encountered in general practice are not caused by local irritation of the nerve ends in the nose, sinuses, teeth or eyes. They more probably result from circulatory changes of some type in the brain, meninges, ganglions, or nerve sheaths." He leaves the controversial subject of "vacuum frontal headache" open. The interesting observation that hypertonic neck muscles cause headaches is thoroughly dealt with. Many theories of histamine and migraine headaches are discussed. Treatment is fully outlined. This book is highly recommended to all rhinologists, residents and graduate students of otolaryngology.

A Manual of Otology, Rhinology and Laryngology. By Howard Charles Ballenger, M.D. Third edition, enlarged and revised. Price $4.50. Pp. 352. Philadelphia: Lea & Febiger, 1947.

This manual of work done on ear, nose and throat is indeed an excellent one for its particular purpose.

The undergraduate should find in it a wealth of accurately and concisely stated fact, without great elaboration. The drawings and photographs in black and white and in color are particularly well chosen. The really common, and hence important, conditions of the ear, the nose and the throat are treated with just the amount of space and scientific endeavor necessary. In addition, this moderate-sized manual should prove a boon to the general practitioner and the assisting nurse for rapid and accurate referral.

Of especial worthiness is the neatness of the text in handling so well important, easily remembered points of etiology, diagnosis, anatomy, therapeutics and prognosis. In short, there is a tremendous amount of useful knowledge packed into this small manual based on an encyclopedic larger volume. The book should prove extremely valuable as an outline of instruction in otolaryngology on an undergraduate plane.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman. Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Chicago. Time: June 21-25, 1948.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Millard F. Arbuckle, 539 N. Grand Blvd., St. Louis.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Old Homestead, Va. Time: April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 15 Whiting Rd., Wellesley Hills, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Chalfonte-Haddon Hall, Atlantic City. Time: April 7-9, 1948.

SECTIONS:

Eastern.—Chairman: Dr. J. Winston Fowlkes, 12 E. 63d St., New York 21.
Place: New York. Time: Jan. 16, 1948.
Southern—.Chairman: Dr. William A. Wagner, American Bank Bldg., New Orleans.
Place: New Orleans. Time: Jan. 23, 1948.
Middle.—Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Place: Columbus, Ohio. Time: Jan. 19, 1948.
Western.—Chairman: Dr. Meade Mohun, 23–2d Ave., San Mateo, Calif.
Place: San Francisco. Time: Jan. 31-Feb. 1, 1948.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Place: Old Homestead, Va. Time: April 12-13, 1948.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

THE ARCHIVES OF OTOLARYNGOLOGY is published by the American Medical Association to stimulate research in the field of otology and laryngology and to disseminate knowledge in this department of medicine.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. George M. Coates, Chief Editor, 1721 Pine Street, Philadelphia 3, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF OTOLARYNGOLOGY. Manuscripts must be type written, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones will be supplied by the Association when the original illustrations warrant reproduction and when their number is not considered excessive.

Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

Matter appearing in the ARCHIVES OF OTOLARYNGOLOGY is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the ARCHIVES OF OTOLARYNGOLOGY or in any of the other publications issued by the Association will not be permitted.

The ARCHIVES OF OTOLARYNGOLOGY is issued monthly. The annual subscription price (for two volumes) is as follows: domestic, $8.00; Canadian, $8.40; foreign, $9.00, including postage. Single copies are 85 cents, postpaid.

Checks, money orders and drafts should be made payable to the American Medical Association.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (three volumes): domestic, $12.00; Canadian, $13.50; foreign, $16.00. Single copies, 35 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $9.00. Single copies, 85 cents.

AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. Includes carefully prepared reviews, based on recent pediatric literature, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $10.00; Canadian, $10.40; foreign, $11.50. Single copies $1.00.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and abstracts of the literature on these two subjects, transactions of the important dermatologic societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): domestic, $10.00; Canadian, $10.40; foreign, $11.00. Single copies, $1.00.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic $10.00; Canadian, $10.40; foreign, $11.50. Single copies, $1.00.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): domestic, $10.00; Canadian, $10.40; foreign, $11.00. Single copies, $1.00, except special numbers.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of pathology. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents, except special issues.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): domestic, $10.00; Canadian, $10.40; foreign, $11.00. Single copies, $1.00.

OCCUPATIONAL MEDICINE—Monthly. Devoted to the advancement of knowledge on the diseases of industry and to the publication of scientific investigation in this field. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00, including postage. Single copies, 75 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: domestic, $15.00; Canadian, $17.00; foreign, $17.00.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **CHICAGO 10**

CONTENTS

PAGE

THE QUESTION OF PROTHROMBINOPENIC HEMORRHAGE FROM POST-TONSILLECTOMY USE OF CHEWING GUM CONTAINING ACETYLSALICYLIC ACID. George S. Livingston, M.D., Chicago, and Edward R. Neary, M.D., Newark, N. J. 1

PLASTIC REPAIR OF THE OBSTRUCTING NASAL SEPTUM. Samuel Fomon, M.D.; Joseph G. Gilbert, M.D.; A. Gilbert Silver, M.D., and Victor Royce Syracuse, M.D., New York 7

CHEMICAL MENINGITIS FOLLOWING USE OF TYROTHRICIN: A CLINICAL AND EXPERIMENTAL STUDY. Frank J. Otenasek, M.D., and David Fairman, M.D., Baltimore 21

NONVIBRATORY TINNITUS: FACTORS UNDERLYING SUBAUDIBLE AND AUDIBLE IRRITATIONS. Edmund Prince Fowler, M.D., New York 29

NASAL HEMORRHAGE: STUDIES OF ASCORBIC ACID, PROTHROMBIN AND VITAMIN K. Harry Neivert, M.D., and Recha Engelberg, Ph.D., New York, and Leo A. Pirk, Ph.D., Nutley, N. J. 37

CONGENITAL LARYNGEAL WEB: ITS ERADICATION. David L. Poe, M.D., and Paul S. Seager, M.D., New York 46

MORPHOLOGIC DEFORMITIES OF THE LOWER LATERAL CARTILAGES. Jacob Daley, M.D., New York 49

CASE REPORTS:

CALCIFIED HEMATOMA OF THE OROPHARYNX SECONDARY TO A GUNSHOT WOUND. Edward H. Diamond, M.D., and Charles W. Perkins, M.D., Norwalk, Conn 64

FIBROMA OF THE TRACHEA. John S. Knight, M.D., M.Sc. (Med.), and Williston P. Bunting, M.D., Kansas City, Mo 67

CLINICAL NOTES; NEW INSTRUMENTS AND TECHNICS:

A SUGGESTION FOR PACKING THE NOSE FOR ANESTHESIA. Major J. Jacob, Indian Army, Medical Corps 71

PROGRESS IN OTOLARYNGOLOGY. SUMMARIES OF THE BIBLIOGRAPHIC MATERIAL AVAILABLE IN THE FIELD OF OTOLARYNGOLOGY:

THE PARANASAL SINUSES (TO BE CONCLUDED). Samuel Salinger, M.D., Chicago 72

ABSTRACTS FROM CURRENT LITERATURE 103

BOOK REVIEWS 110

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES 112